Assessment and management of acute poisoning by petroleum products

FK Seymour and JA Henry*

Academic Department of Accident and Emergency Medicine, Imperial College School of Medicine, St. Mary’s Hospital, Queen Elizabeth the Queen Mother Wing, South Wharf Road, London W2 1NY, UK

Petroleum products are highly complex chemical mixtures consisting predominantly of hydrocarbons. Their composition varies with source and intended use of the product. Virtually all are blended products that come into contact with man in a wide range of circumstances. Their toxicity for man is generally low and the use of additives rarely affects the toxicity of the final product. Because products are blended to meet performance, and not chemical specifications, their composition varies significantly. Management of toxicity benefits from simplified guidelines that consider the product by its type. Management in most cases is symptomatic, but the doctor needs to be aware of the potential for development of sequelae such as aspiration pneumonia and central nervous system (CNS) depression.

Local and systemic effects of exposure to hydrocarbons are reviewed, as are immediate assessment and recommended management of acute exposure to petroleum products. Because of the large scope of this subject, this paper limits itself to acute toxicity of petroleum products encountered in the public domain. It does not address topics such as chronic toxicity, solvent abuse, petrochemicals, or pesticides. Human & Experimental Toxicology (2001) 20, 551–562.

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Introduction

Petroleum products find their way into most people’s lives, refined or derivatized as a variety of fuels, lubricants, household and industrial products, medicines and plastics. The term ‘petroleum products’ is here used to cover those products manufactured in a petroleum refinery or blending plant that are available in the public domain. Examples are petrol, heating or diesel oil and lubricants. It does not include raw materials for manufacture of related products such as benzene and toluene, chlorinated and oxygenated solvents, pesticides, or other specialty chemicals. It also excludes ‘forecourt products’ such as antifreeze, screen washer and brake fluid.

Exposure to petroleum products mostly results from product use but may occasionally occur due to accidents and failures during their manufacture, transportation, use and disposal. Some major problems are well known and have been, or are being, reduced or eliminated. The well-known examples of toxicity due to tetraethyl lead and tetramethyl lead in petrol have been eliminated by the exclusion of these products in the European Union and other areas. Other hazards remain, although in most cases exposure is preventable and, hence, risk can be effectively eliminated. The most important clinical problems are the central nervous system (CNS) depressant effect of volatile petroleum products and pulmonary aspiration of low-viscosity products. Prolonged exposure to high concentrations of petroleum products by inhalation may cause CNS deterioration. Cutaneous sensitization and irritation are often regarded as likely, but, in fact, are unusual. Defatting of skin can be a problem in chronic exposure situations where good handling practices are not enforced. We concentrate on the medical assessment and management of incidents involving acute exposure. Chronic toxicity is rare, and such problems as may arise are more fully dealt with in textbooks of occupational medicine.

Petroleum refining

The refining of crude oil is a complex procedure. A brief outline is given in Appendix A. The properties of basic petroleum products are determined by crude oil source, boiling range, process history and other factors. Finished products consist almost entirely of hydrocarbons (paraffins, cycloparaffins, olefins, aromatic hydrocarbons, and others) and usually contain additives. Various types of additives may be used but they are rarely present at concentrations that affect the acute toxicity of the product or the treatment of poisoning.

*Correspondence: JA Henry, Academic Department of Accident and Emergency Medicine, Imperial College School of Medicine, St. Mary’s Hospital, Queen Elizabeth the Queen Mother Wing, South Wharf Road, London W2 1NY, UK

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Information sources and resources

Much of the literature on acute hydrocarbon exposure dates back as far as the 1970s. Since that time, improved safety and preventative measures have markedly reduced the number of severe exposure cases. This is also apparent from a consideration of the world literature on the topic, which is largely limited to individual case reports.

Poisons centres originated over 50 years ago in the USA as a response to the problem of acute poisoning, particularly in children, in the home. They were established so that the composition, toxicity, and action to be taken in case of accidental exposure of small children to household products could be relayed immediately to the enquirer. Their role has expanded greatly since then. Poisons centres are increasingly involved in advising on the environmental toxicity of products, industrial poisoning and in planning for chemical emergencies. Many poisons centres provide laboratory analysis for suspected toxins and facilities for the treatment of poisoned patients. Poisons centres have always required product information in sufficient detail to maintain an up-to-date database for the provision of medical information and advice.

Petroleum products are manufactured to a performance specification, not a chemical one. Hence the chemical composition of any product type is generally variable as well as extremely complex (e.g., a gasoline or fuel oil will typically contain well over a hundred different chemicals). Knowledge of the precise chemical composition of petroleum products is of virtually no value in the treatment of poisoning.2–4

The generic terms determined by the EU Commission, with some associated definitions, are fully sufficient to ensure that proper medical treatment is not compromised. These product names are included in European Inventory (EINECS) and used in Annex I (the list of dangerous substances) of the EU’s Dangerous Substances Directive (Directive 67/548/EC).

Although the issue of confidentiality may rise with respect to additives present in petroleum products, this is rarely of concern to Poisons Centres.

In fact, knowing the systematic chemical names of such materials is more than likely to confuse the identification and management of toxicity. An indication of any physiologically active constituents, combined with risk and safety phrases, is sufficient in nearly all cases. Such information is available through an internationally recognized nomenclature system used on additive labels and data sheets — the major means of providing information for users and health professionals, respectively. This system, which has been in use for about 20 years, is fully documented in the TOMES Plus INFOTEXT Information System published by MICROMEDEX, and held by poisons centres around the world.

Health and safety legislation relating to the workplace requires communication of information on the prevention and management of industrial accidents. Computer programmes, websites and published handbooks are at the disposal of health professionals. CONCAWE (the oil companies’ European organization for environment, health and safety, established in 1963) has produced numerous reports providing information and guidance on petroleum products, ranging from first-aid advice to detailed product dossiers. They are well researched and referenced and are available on request.5 An example of the first-aid advice available for exposure to diesel/gas oil is given in Appendix B.

Prevention

Petroleum products used in the domestic environment are still frequently stored in the home or garage in unmarked containers or beverage bottles. They may also have attractive aromas and be brightly coloured. It is not surprising therefore that most cases of exposure in the home involve accidental ingestion by young children. Whilst education in schools is beneficial, a large proportion of childhood poisoning occurs in the preschool group. Children under 6 accounted for more than 50% of all exposures reported to poisons centres in the USA in 1996, of which 2.4% involved hydrocarbons.6 Parents should be educated to keep potentially toxic materials out of the reach of children. Familiar bottles or containers should not be used for the storage of these products. The companies supplying the domestic market with small quantities of products also have a role to play in developing suitable packaging and labelling of their products. The introduction of child-resistant containers, often required by law, has raised public expectation of suitably safe packaging and presentation not only of medicines but also of all household and DIY products. Under current EU legislation, both manufacturers as well as users are perceived to have some responsibility for ensuring the safe storage of their products in the home.

Whilst steps can be taken to prevent the accidental ingestion of petroleum products by children, a different approach is required against the deliberate inhalational abuse of hydrocarbons occurring typically in adolescents. There are about 150 deaths each year in Britain from this cause.7,8

As a result of increasing concerns and the gradual development of regulatory controls, industrial poisoning is rare nowadays. The precautions recommended to minimize adverse health effects are all aimed
at limiting exposure. The onus is on both the manu-
ufacturer and user to assess risks, and implement work
place standards. Fuels are normally stored and
handled in systems which, for the most part, are
‘closed’. Hence, for most of the time, exposure and,
thus, any risk of adverse effect is kept very low.
However, during certain operations such as loading
or unloading or the maintenance of storage tanks, it is
appropriate to take special care to avoid skin contact
and excessive inhalation of vapour.

**Physiologically active constituents**

Consideration of any petroleum product should in-
clude the two main groups of constituents: the hydro-
carbon fraction and the additives. With the exceptions
of tetraethyl lead and tetramethyl lead, which are no
longer allowed as fuel additives in the European Union
and many other areas, additives are unlikely to affect
the clinical features of hydrocarbon toxicity.

**Hydrocarbons**

Hydrocarbons contain hydrogen and carbon atoms
only. Their health effects vary, inter alia, with their
volatility and viscosity (Table 1).

Volatility describes the tendency of a liquid to
vaporize. Generally speaking, the lower the molecular
weight and the higher the degree of unsaturation or
aromaticity, the greater the volatility. Some hydro-
carbons are gaseous at ambient temperatures. The
more volatile hydrocarbons are more lipid soluble
and therefore more readily absorbed by inhalation or
ingestion. They more readily enter the CNS and other
target organs.

Viscosity is the resistance offered by a fluid to flow.
It is also an indirect measure of molecular size. It is
the single most important physical property influenc-
ing the aspiration tendency of a liquid. The viscosity
of a substance determines not only the likelihood of
its entry into the trachea, but also the rate and extent
of its penetration into the terminal bronchioles and
alveoli. The lower the viscosity, the greater the risk of
aspiration.

**High volatility, minimal viscosity**

- simple gases, e.g., methane, propane, butane and
pentane;
- light aromatic hydrocarbons, e.g., benzene and
toluene.

Inhalation of these substances can replace alveolar
gas, causing hypoxia. In addition, they can easily
cross the alveolar-capillary membrane and cause CNS
symptoms. The lung is spared injury, but cardiotoxic
effects have been reported. Gastrointestinal absorp-
tion can be significant.

**Intermediate volatility, low viscosity**

- e.g., petrol, naphtha and hydrocarbon solvents.
The primary problem with these agents is aspiration.
However, they may cause CNS depression when
inhaled. Effects from gastrointestinal absorption are
not significant.

**Low volatility, low viscosity**

- e.g., diesel and heating oils. The main problem
with these compounds is aspiration pneumonia.
Gastrointestinal absorption is minimal.

**Minimal volatility, high viscosity**

- e.g., lubricating oils, mineral oil bitumen. These
materials are highly viscous, essentially non-toxic,

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**Table 1  Hydrocarbons and their characteristics**

<table>
<thead>
<tr>
<th>Substance</th>
<th>Synonyms</th>
<th>Uses</th>
<th>Composition/viscosity*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Petrol</td>
<td>Petroleum spirit; gasoline; naphtha</td>
<td>Motor fuel</td>
<td>C_{4}/C_{12} mixed hydrocarbons and alcohols. SSU&lt;35</td>
</tr>
<tr>
<td>Paraffin</td>
<td>Kerosene/kerosine; barbecue lighter fluid; jet fuel; lamp oil; No. 1 fuel oil</td>
<td>Heating/cooking fuel</td>
<td>C_{9}/C_{16} paraffinic hydrocarbons. SSU&lt;35</td>
</tr>
<tr>
<td>Diesel fuel/gas oil</td>
<td>Automotive gas oil; DERR; domestic fuel oil; heating oil; industrial gas oil; marine diesel; No. 2 and No. 4 fuel oil.</td>
<td>Fuel; industrial solvent</td>
<td>C_{11}/C_{25} mixed hydrocarbons. SSU&lt;60</td>
</tr>
<tr>
<td>Lubricating oil</td>
<td>Various branded engine oils and light oils for domestic use</td>
<td>General and specific lubrication</td>
<td>C_{15}/C_{25} mixed hydrocarbons and additives. SSU&gt;100</td>
</tr>
<tr>
<td>Fuel oil</td>
<td>Light/medium/heavy fuel oil; marine fuel oil; No. 6 fuel oil; Black Oil; residual fuel</td>
<td>Industrial and marine engines; industrial heating plant; power generation</td>
<td>Very complex mixtures of hydrocarbons from about C_{20} upwards SSU 60–6000</td>
</tr>
</tbody>
</table>

*SSU (Saybolt seconds universal) is a common measure of viscosity, in efflux time in seconds.
but may cause lipoid pneumonias in cases of direct aspiration. This form of pneumonia is more localized and less inflammatory than that produced by lower viscosity products.\textsuperscript{9,10}

It should be noted that hydrocarbons such as toluene and xylene are sometimes used as solvents in pesticides, which are widely available, especially in developing countries. Although most of the toxicity is usually caused by the pesticide there may be significant toxicity from the solvents.

\textit{Local and systemic effects of hydrocarbons}

Local effects depend on the part of the body exposed, while systemic effects relate both to the amount absorbed and to the degree of pulmonary or other organ toxicity caused.

\textit{Local effects} Cutaneous injury appears to be due to irritant effects and fat solvency properties of hydrocarbons. It is mainly due to the removal of fat within the layers of the skin, hence the term ‘defatting’. Dehydration of the skin probably also contributes to the damage. The depth of injury is related to the duration of exposure and the concentration of the agent, resulting most commonly in superficial damage. The affected area may be erythematous or blistering may occur. A severe burn appears red and raw, often with a margin of dead skin at the edges. Cutaneous absorption of petroleum products may occur, but with acute exposures its clinical significance is probably negligible.\textsuperscript{11}

Ocular exposure usually causes little or no injury. There may be considerable stinging and discomfort. Photophobia, redness and transient corneal irritation may be present.

Pulmonary pathology results most commonly from aspiration into the bronchial tree of hydrocarbons with low viscosity. Aspirated hydrocarbons disrupt surfactant and the bronchial epithelial cell barrier, leading to alveolar instability, early distal airway closure, ventilation/perfusion mismatches and subsequently hypoxaemia.\textsuperscript{12} Initially cyanosis may result from replacement of oxygen by vaporized hydrocarbons. Subsequent hypoxaemia occurs from surfactant loss and direct alveolar injury. Bronchospasm may contribute to ventilation/perfusion mismatches.

Gastrointestinal effects, although unpleasant, are usually only transient and result from direct local irritation of the pharynx, oesophagus, stomach and small intestine. Patients may experience symptoms such as nausea, vomiting and diarrhoea. Small areas of fatty infiltration and congestion of the liver have been described, and a single fatality has been reported in an abuser of butane aerosols.\textsuperscript{13}

\textit{Systemic effects} The CNS toxicity occasionally observed following hydrocarbon exposure appears to be indirect and secondary to the pulmonary involvement. Experiments using paraffin in baboons suggest that the primate brain is resistant to the direct effects of kerosene; and that the most potent cause of CNS damage is hypoxia secondary to pneumonitis.\textsuperscript{14} Neurological abnormalities include behavioural changes, movement disorders (resting and action tremor, myoclonus, chorea, ataxia) as well as pyramidal signs and seizures.

Cardiac manifestations of hydrocarbons are thought to be responsible for numerous reports of sudden death with abuse by inhalation. The mechanism by which dysrhythmias occur is believed to be sensitization of the myocardium to endogenous catecholamines.

Glomerulonephritis, renal tubular acidosis and chronic tubulointerstitial nephritis have all been associated with long-term hydrocarbon exposure. Intravascular haemolysis following petrol aspiration has been reported in a small number of cases, possibly due to damage to the red blood cell membrane and induction of lipolysis by lipid solubilization.\textsuperscript{15} Petrol also contains a small amount (<1%) of benzene, which is a human carcinogen.

\textit{Additives} A wide range of additives and other chemicals is used in fuel products and lubricants, some of which are classified as ‘dangerous’.\textsuperscript{9} However, lubricants excepted, these chemicals are generally used at such low concentrations that they have no impact on the toxicity of the finished products. Finished lubricants may contain as much as 15–20% of additives, but these are mostly not classified as dangerous or, again, are used at such concentrations that the lubricant toxicity is not significantly affected. The additives themselves are commonly manufactured in or diluted with oil products of various kinds and these may have a significant effect on the way the additive is classified and labelled. Additives containing a significant proportion of a base oil will, if ingested, cause some irritation of the digestive tract and associated symptoms (nausea, vomiting and diarrhoea); with additives containing a significant level of a low-viscosity oil product, there is a danger, should vomiting occur, of aspiration into the lungs causing chemical pneumonitis. A more detailed discussion of additives and their toxicity can be found elsewhere.\textsuperscript{16}

\textsuperscript{9}The rules for classification and labelling of dangerous chemicals within the EU will be found in Directive 67/548/EC as amended, and its annexes. The classes of danger cover health effects (very toxic, toxic, harmful, corrosive, irritant, sensitizing, carcinogenic, mutagenic and toxic for reproduction), as well as physicochemical hazards and environmental effects.
Diagnosis and assessment

Triage

Petroleum product toxicity can lead to a wide range of presentations, from accidental exposure in children to industrial accidents. In addition, there are various ways in which petroleum products may cause toxicity.

Most patients who have ingested hydrocarbons and who are asymptomatic can be managed at home provided someone responsible is able to observe them. There must be no history of specific toxic components involved, which is generally the case with most commercially available products, such as fuels, solvents and cleaning agents. The patient must have rapid access to hospital in case delayed symptoms develop.

All patients who have had symptoms suggestive of aspiration (choking, coughing, gagging) should be assessed in the emergency department. By the time that they come to medical care, most of these are asymptomatic. Asymptomatic patients should have a chest X-ray and be observed for 6 h. They can usually be discharged if no abnormality develops.

The main indications for admission include:

- Respiratory signs or symptoms, fever or lethargy. These usually appear soon after ingestion.
- Ingestion of systemically absorbed substances. Patients should be observed for signs of CNS toxicity.
- Suicide attempt, requiring psychiatric assessment.
- Ingestion may be a presenting feature of child abuse. Although very uncommon, this is a diagnosis not to be missed, and should always be considered when any child presents with toxic ingestion.

Figure 1 summarizes these recommendations.

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Figure 1 Algorithm for management of acute hydrocarbon ingestion. ABC: attention to airway, breathing and circulation; CXR: chest radiograph.
History

A detailed history should be taken from the patient or if necessary from witnesses. In childhood poisoning it is often difficult to know whether the patient has ingested the suspected substance or not. Key information required is normally as follows:

- Age
- Substance(s) involved or suspected (The container with its label should be brought with the patient. A sample of the product is not usually required as there is little need to analyse the substance. The exception to this is when substances have been stored in inappropriate containers, and composition is not known.)
- Time of exposure and duration of exposure
- Mode of exposure: oral, inhaled or ingested
- Has there been any contact with skin or eyes?
- Has the patient vomited or had any other symptoms?
- Has the patient taken any alcohol or other drugs that might affect the clinical presentation?
- Past medical history and current drug treatment

Immediate management

ABC

Clinical history and assessment. Fluid administration should be aimed at replacement of losses while taking care to avoid precipitating pulmonary oedema by overgenerous hydration.

Respiratory assessment

A patent airway is the first priority. If there is evidence of respiratory involvement, a high inspired oxygen concentration should be provided and nebulized bronchodilators may be used with caution if bronchospasm is severe (see below under drug treatment). The next consideration is to decide whether the patient requires mechanical ventilation. Signs of respiratory distress such as cyanosis, retraction, and the rate and depth of respiration should be noted. If there is any doubt, the oxygen saturation and minute volume should be measured, and if the minute volume is less than 4 l/min in an adult, endotracheal intubation and mechanical ventilation are likely to be required. Auscultation may demonstrate crepitations, crackles, wheezing and diminished breath sounds, but abnormal findings are often absent. It is important to remember that lower airway involvement may be present despite a normal chest examination. A typical hydrocarbon odour is often detected in these patients but is not a reliable indicator of significant ingestion or aspiration.

Most patients will become symptomatic within 30 min of ingestion. Almost immediately upon aspiration there are signs of tracheobronchial irritation, manifested as coughing and choking. These signs may be transient due to the initial volatilization of the petroleum distillates. Aspiration into the lungs is indicated by gasping and more prolonged coughing.

Arterial blood gases should be measured in the seriously unwell patient. Patients presenting with significant respiratory compromise need an urgent chest X-ray, whereas patients with minimal symptoms should be reassessed over a 6-h period to see if an X-ray is indicated.

Signs and symptoms may progress over the first 24 h. Initially, most patients with significant exposure become hypoxaemic from hyperventilation. Nasal flaring, intercostal retractions, dyspnoea, tachypnoea, and varying degrees of cyanosis may follow. In severe cases, pulmonary oedema and haemoptysis or pink frothy sputum may be evident, later leading to shock and cardiorespiratory arrest. Derangement of normal pulmonary surfactant properties by aspirated hydrocarbons has led to the suggestion that early use of continuous positive-airway pressure (CPAP) or positive end-expiratory pressure (PEEP) may be beneficial. Children with severe pulmonary complications should be considered for treatment. Studies have shown that treatment decreases barotrauma, air leaks and morbidity. Extracorporeal membrane oxygenation has occasionally been used to oxygenate the patient’s blood whilst allowing lung tissue to heal. It consists of a modified cardiac bypass procedure, but does not require the chest to be opened.17 If death is going to occur, it usually does so within the first 24 h.18 Otherwise, pulmonary symptoms plateau in about 48 h, with complete resolution in about 3 to 5 days.

Intravenous exposure, though rare, usually causes a chemical pneumonitis, occasionally haemorrhagic in nature.

Cardiovascular assessment

Pulse rate and blood pressure should be recorded and intravenous access secured. Patients with more than mild symptoms should be monitored by pulse oximetry and an electrocardiogram should be performed. Myocardial involvement is rare after acute hydrocarbon ingestion. However, dysrhythmias can occur during solvent abuse. The mechanism is believed to be a result of hydrocarbon sensitization of the myocardium to endogenous catecholamines.19 Sudden death has been reported, especially with abuse of chlorinated and fluorinated hydrocarbons.20,21
Skin and eye assessment

After spillages which have caused skin contact, contaminated clothing should be removed and the affected area washed with soap and plenty of water. Accurate assessment of the burn surface area and proper fluid management may be required. Contact with petroleum distillates can cause a variety of skin manifestations ranging from mild erythema to full-thickness skin loss. Features of eczemoid dermatitis such as redness, itching and inflammation may be seen. A clinical picture resembling toxic epidermal necrolysis with areas of bullae and denuded skin has been described. Compared to thermal and other chemical burns, the latency of the signs and symptoms may be considerably longer.

Subcutaneous injection of hydrocarbons may lead to cellulitis and abscess formation. Initial treatment consists of cleansing the wound area, tetanus prophylaxis and using X-rays or ultrasound to determine the location of material. High-pressure injection injuries (e.g., as arising from pinhole leaks in high-pressure hydraulic equipment) require particular care, though initially there may be no signs or symptoms. Significant swelling and pain can develop within a few hours. Sterile abscess formation, ischaemia and chronic bone and connective tissue injuries occur. Surgical debridement and irrigation is the recommended treatment for any significant high-pressure injection injury. This is beyond the scope of the present review.

In the case of eye exposure to volatile substances, it is more appropriate to hold the lids open in order to allow the substance to evaporate rather than to wash out the eye with water. Ocular exposure generally causes little or no injury provided there are no abrasions. Considerable discomfort, photophobia, redness and transient corneal irritation may be present, but resolve on conservative management.

Gastrointestinal assessment

Gastrointestinal symptoms following hydrocarbon ingestion are common, but are usually minor. Turpentine is particularly associated with such symptoms. There may be local irritation of the mouth and pharynx. In more severe cases, hydrocarbons can cause nausea, vomiting, abdominal pain and distension. Diarrhoea, haematemesis and melaena are rare.

The ability of hydrocarbons to produce spontaneous vomiting is associated with a high risk of developing aspiration pneumonitis. Any vomit produced should be inspected for evidence of ingestion, and a 20-ml sample saved in a universal container.

Gastrointestinal decontamination The traditional methods of gastrointestinal decontamination have been closely reviewed recently, leading to the production of position statements from the American and European Toxicology associations. Unfortunately these statements are of little use in the management of hydrocarbon ingestion, because of the paucity of data and the special properties of hydrocarbons. The situation may be briefly reviewed as follows:

Emesis Although syrup of ipecacuanha is the only substance now available for inducing emesis, it is not recommended for ingestion of volatile hydrocarbons because it may lead to pulmonary aspiration.

Gastric lavage Gastric lavage can only be carried out after endotracheal intubation to protect against aspiration. This reduces its use in practical terms to the patient who is already extremely ill or unconscious.

Activated charcoal Activated charcoal does not effectively adsorb petroleum distillates. However, it may need to be given to adsorb a toxic substance formulated in a petroleum base.

Catharsis Cathartics are contraindicated in all toxic ingestions.

Whole bowel irrigation There is currently no evidence that whole bowel irrigation is of benefit in the ingestion of hydrocarbons.

The risk of aspiration posed by attempted gastric emptying is greater than the potential benefit in most cases.

Neurological assessment

Studies show that gastrointestinal absorption of hydrocarbons does not cause pulmonary pathology or major CNS toxicity. However, inhalation of hydrocarbons may be associated with signs of CNS toxicity. This may be due to a direct effect on the CNS or secondary to hypoxia. The conscious level should be recorded using a coma grading such as the Glasgow Coma Scale. Patients whose altered mental status may also be due to alcohol or drug misuse should receive naloxone, glucose and thiamine as appropriate. Hydrocarbon exposure results in CNS depression; somnolence, dizziness and hyporeflexia are common. It is very unusual for convulsions and coma to occur. Unlike the aliphatics, systemically absorbed aromatic and halogenated compounds may have
marked initial excitatory effects leading to euphoria, agitation, delirium, hyperreflexia and seizures.

Other organ systems
Transient hepatosplenomegaly and renal and hematological abnormalities may occur, depending on the exposure and the substance. Since petroleum distillates are used as the base for organophosphate preparations the breath odour of a volatile substance may mask the true diagnosis. A cholinergic crisis must be considered if symptoms such as profuse salivation, lacrimation, diarrhoea, bronchorrhoea, cramps, miosis and urinary incontinence are present.

Investigations
In straightforward cases of hydrocarbon exposure, there is usually no point in routinely ordering such tests as full blood count, urea and electrolytes, liver function tests, chest X-ray and electrocardiogram. However, in the patient who is ill enough to require admission to hospital, a small number of baseline tests are indicated as deterioration may occur.

In addition to continuous monitoring of oxygen saturations, arterial blood gases must be monitored in all patients with respiratory symptoms. Varying degrees of hypoxia without hypercapnia are the most common findings. Occasionally a metabolic acidosis is seen (e.g., with toluene).

The full blood count may show a leukocytosis with left shift, a relatively early finding which may persist for several days. Serum electrolytes, coagulation studies, urinalysis, and renal and liver function tests are usually only abnormal in cases of chronic exposure.

Measurement of blood hydrocarbon levels has no place in acute diagnosis or management and is of little value except for forensic, medicolegal or research purposes. However it is normal practice to save an anticoagulated blood and urine sample on admission in case analysis should subsequently be necessary.

Chest X-rays form the mainstay of radiographic studies. Chest X-ray abnormalities correlate poorly with clinical symptoms, and may be abnormal in patients who are asymptomatic. Of patients hospitalized with suspected hydrocarbon aspiration, 70–75% have chest X-ray signs. Abnormalities may start to appear within 30 min but more commonly occur later, and may not develop for up to 24 h after exposure. The most commonly seen abnormalities are increased bronchovascular markings and bilateral basal shadowing. Upper lobe signs are uncommon. Infiltrates usually reach their maximum in 3 to 4 days and generally clear within 2 weeks of onset. Other rare findings include pleural effusion, pneumothorax and pneumomediastinum.

Drug treatment
Corticosteroids
The evidence suggests that corticosteroids are ineffective in altering the acute course of hydrocarbon pneumonitis. Although a single case report of a late-treated case suggests that high-dose corticosteroids may produce resolution of lung damage, the evidence from animal studies of combined steroid and antibiotic therapy have not demonstrated any beneficial effect. In fact, corticosteroid suppression of the immune response may even promote bacterial colonization. There is no evidence that bacteria play a role in the pathogenesis of chemical pneumonitis.

Superinfection
Bacterial superinfection may be difficult to diagnose. Fever and leukocytosis are common in uncomplicated hydrocarbon pneumonitis, and bacterial superinfection is therefore a microbiological diagnosis. Prophylactic antibiotics should not be prescribed routinely.

Bronchodilators
Bronchospasm may exacerbate respiratory distress, and it is tempting to give bronchodilators to wheezy patients, but they should be used with caution. A sympathomimetic agent such as salbutamol may be used if there is a decrease in forced expiratory volume, as the risk of precipitating dysrhythmias is negligible. Epinephrine should be avoided because of the potential risk of myocardial sensitization to catecholamines.

Long-term effects
Full recovery is the normal outcome of hydrocarbon poisoning. Some researchers have suggested that minor abnormalities of pulmonary function may persist, but the clinical significance of these is not clear.

Chronic exposure and carcinogenicity
Benzene is considered a human carcinogen. Acute myelocytic and monocytic leukaemias have been reported with chronic benzene exposure, as well as lymphoproliferative disorders. Chemicals containing straight chains of six carbon atoms (e.g., n-hexane...
and methyl-n-butyl-ketone) are also known for their potential to cause peripheral neuropathy. Toluene inhalation over long periods of time has been associated with renal tubular acidosis and peripheral sensorimotor neuropathy. Reviews of chronic toxicity and carcinogenicity are outside the scope of this review but are available.

Conclusion

Every clinician needs a basic awareness of the implications of acute exposure to petroleum products, especially as the clinical presentation may be acute, requiring urgent action. Once the initial emergency has been dealt with, there are a large number of reliable information sources that can be consulted to provide data on the composition, clinical features and management of individual problems. Many of these are based on a general knowledge of the chemical properties and potential toxicity of the substance concerned, as the literature is relatively sparse. The paucity of clinical experience also shows that preventive measures have been increasingly successful. Hopefully, petroleum products should form a decreasing proportion of the emergency physician’s workload, but preparedness is needed for the cases that will still inevitably occur.

Acknowledgement

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References

Appendix A

The refining of crude petroleum oil

The aim of petroleum refining is to convert crude oil into marketable products. These range from liquefied petroleum gases (LPG), through petrol (gasoline), jet fuels, the so-called distillate products (such as diesel fuel, paraffin and heating oil) and lubricant base stocks, to residual products like bitumen and heavy fuel oils. Crude oils vary widely in the yields and qualities of the streams obtained by primary distillation. Hence many of these first cuts require further treatment if the final (usually blended) products placed on the market are to meet required technical and environmental standards. Accordingly, refineries vary greatly in complexity and in terms of their processing units.

After electrostatic removal of water and salt, crude oil is distilled at atmospheric pressure into a number of fractions according to boiling range. The maximum temperature in this stage is about 360°C. Above this temperature, hydrocarbons start to ‘crack’ (thermally decompose). Raw gases, gasoline components, paraffin and heating/diesel oil steams are obtained at this stage. The residue from atmospheric distillation is then distilled under vacuum to yield lubricant base stocks, heavy gas oil and residual products. The residue from this second distillation may be thermally cracked to produce more blending components for various fuels.

Most refineries also operate some secondary treatment plants. These are generally designed to improve product quality and/or convert lower value streams into a range of more valuable ones. Product quality is commonly improved by: 1) removal of malodorous and toxic mercaptans by oxidation to odourless, low-toxicity disulphides or 2) removal of noxious sulphur compounds by hydrogenation. Common upgrading processes include catalytic cracking, hydrocracking, visbreaking, coking and severe thermal cracking. All of these processes convert a high proportion of the larger molecules into smaller, higher-value ones. Other important upgrading processes include chemical rearrangement by catalytic reforming, isomerization and alkyla

en. In reforming and isomerization, virgin (untreated) naphthas of low octane number are converted to high-octane components of petrol. In alkyla

tion, smaller molecules are combined into larger ones, which are more valuable as gasoline components. Upgrading processes commonly increase toxicity because the olefinic, branched-chain and aromatic hydrocarbons so formed are generally more toxic than their predominantly straight-chain paraffinic precursors.

The majority of marketed petroleum products are blends of a number of streams. The exact compositions are calculated so that the final product meets concurrently the performance, quality and environmental standards required. Petrol commonly contains up to 10 or more components, each of which is a mixture of many closely related hydrocarbons.

References

While fuel oils will often contain even more components, distillate fuels usually contain fewer. Lubricants are blended from various base stocks, mostly produced by vacuum distillation. Top-grade lubricants must contain a high proportion (10–20%) of additives in order to meet today’s severe performance requirements. Generally speaking, the additives, even at these high concentrations, have very little impact on the toxicity of the products as marketed.

Appendix B

Extract from First Aid Emergency and Medical Advice CONCAWE report 1/97 (ref. 5)

CONCAWE report no. 1/97

DIESEL FUEL/GAS OIL

Synonyms Automotive gas oil (AGO), DERV, diesel, domestic fuel oil, gas oil, heating oil, industrial gas oil (IGO), light vacuum distillate, marine diesel, No. 2 fuel oil, No. 4 fuel oil.

Description Gas oils and diesel fuels are hydrocarbon mixtures. Gas oils may also be marketed as industrial solvents.

EMERGENCY TREATMENT

Warning: Spillages make surfaces slippery.

Inhalation This is unlikely because of the low vapour pressure of these products at ambient temperatures. Exposure to vapours may however occur when these products are handled at high temperatures with poor ventilation. If symptoms arise from inhalation of vapours, remove the casualty to fresh air. If the casualty is:

- not breathing, give assisted respiration by the mouth-to-mouth or mouth-to-nose method
- unconscious but breathing, place in the unconscious (recovery) position.

If necessary, give external cardiac massage and obtain medical advice.

Ingestion If gas oil or diesel fuel is ingested, do not induce vomiting but obtain medical attention immediately. [Also see advice on aspiration (page 7)].

Skin contact Where significant skin contact has occurred remove any contaminated clothing and wash all the affected skin areas thoroughly with soap and water. Injection under the skin can occur when using high-pressure equipment. If this has happened the casualty should be sent immediately to a hospital. Even when there are few or no symptoms, do not hesitate to refer to a hospital if injection under the skin is suspected.

Eye contact If the eyes are affected, irrigate them immediately with copious amounts of water. If irritation occurs and persists, obtain medical advice.

DIESEL FUEL/GAS OIL

Synonyms Automotive gas oil (AGO), DERV, diesel, domestic fuel oil, gas oil, heating oil, industrial gas oil (IGO), light vacuum distillate, marine diesel, No. 2 fuel oil, No. 4 fuel oil.

Description Gas Oil is a generic term used to describe complex, variable mixtures of petroleum hydrocarbons, with a carbon number range Of C9–C25. The normal boiling point range is 150–450°C, with a flash point range of 55–80°C. They are manufactured by blending ‘middle distillate’ refinery streams and hence their composition varies depending on the source of crude oil processed and refinery process stream availability. Normally, they are mobile liquids with a characteristic odour. Marketed gas oils may contain small amounts of chemical additives to improve performance and in some cases may be dyed for commercial or tax purposes.

Toxicology/health aspects

In general gas oils are unlikely to cause systemic toxicity following accidental exposure by ingestion, inhalation or skin contact. Accidental ingestion may however cause transient gastrointestinal effects. Skin contact may cause irritation, particularly if contact is frequent or prolonged. Gas oils also have a defatting action on the skin, which can result in drying, cracking and dermatitis. Accidental splashes entering the eye may cause slight irritation and discomfort.
Such effects, however, are only likely to be transient, and permanent damage is considered unlikely. The low vapour pressure of gas oils normally precludes generation of high concentrations of vapour. Use at elevated temperatures, and in applications involving spraying or mechanical aerosolisation, can result in exposure to concentrations of mist or vapour which may cause irritation of the eyes, nose and respiratory tract. Exposure to higher concentrations may cause signs of CNS depression, e.g., headache, dizziness, mental confusion. The main potential health hazard, however, is the possibility of severe, potentially fatal, damage to lung tissue, which can occur following aspiration of even small amounts of liquid gas oil into the lungs. An additional, potentially serious hazard with gas oils, is the possibility of high pressure injection through the skin. This can cause considerable damage to underlying tissues, even when surface effects are minimal.

Principal routes of exposure

In normal use, the main routes of exposure to gas oils are by inhalation and/or skin contact. Although most gas oils are insufficiently volatile to give rise to significant concentrations of mist/vapour at ambient temperatures, higher concentrations may be generated at elevated temperatures, during spraying operations or by fast moving machinery. During handling, accidental skin/eye contact may occur and in some applications prolonged skin exposure is possible. Accidental ingestion of significant quantities of these products is an unlikely event. If it does occur the principal concern is the possibility of aspiration of low viscosity material into the lungs. Direct exposure to gas oils can arise from both industrial and consumer (general public) activities. In addition, exposure may occur indirectly, as a consequence of environmental contamination e.g., from spills or leakages. For the general public, the main potential exposure situation is during automobile refuelling. Exposures are likely to be small, with accidental splashes on the skin, and possibly inhalation exposure the most likely to occur. Accidental ingestion of small volumes may also occur, mainly as a result of attempts to siphon product by mouth. In industrial applications, and following accidental spillage, there is a potential for workers to be exposed to much larger amounts. Skin and inhalation exposure is likely, particularly if gas oils are sprayed, or following a major spillage.

Additives

Gas oil based products, depending upon application, can contain a diverse range of proprietary chemical additives e.g., antioxidants, flow/com bustion improvers, detergents or corrosion inhibitors. Although some of these compounds are acutely toxic, the concentrations found in gas oil products are relatively small; for fuels, individual additives are present typically at <0.1%. In the case of industrial products where the amounts may be up to 10%, the additives are of lower toxicity. Thus, in the case of gas oil products for both fuel and other uses, it is the toxicity of the gas oil which dictates the necessary emergency treatment regimes.

Principal sources of exposure

Information for doctors

Administration of liquid medicinal paraffin or activated charcoal may be used to reduce the speed of absorption through the digestive tract. Gastric lavage should only be done after endotracheal intubation in view of the risk of aspiration which can cause serious chemical pneumonitis.

Gas oils and diesel fuels have an irritating effect on skin and eyes, but normal conditions of storage and use will afford little opportunity for such exposure, although excessive skin contact must be avoided. Frequent or prolonged contact with these products may remove the natural protective fats from the skin and cause irritation and dermatitis.

References: