Miscellaneous Pesticides, Solvents, and Adjuvants

There are a variety of pesticides that do not fall into the broad categories described in other chapters in this manual. Many of them are widely used and are therefore associated with a high probability of human exposure. Some have significant toxicity as well as a likelihood of human exposure, and are of real concern. Many of the solvents and adjuvants used in the formulation of pesticides also present a high likelihood of human exposure. Such exposures can result in significant toxic effects that in many cases exceed the toxicity of the active pesticide ingredient(s). Furthermore, it is sometimes more difficult to obtain information about the solvents and adjuvants, complicating the issues of diagnosis and management.

4-AMINOPYRIDINE

Toxicology

4-Aminopyridine is a highly toxic white powder used as a bird repellent. It works by making one or two birds acutely ill, thus warning off the remaining birds by cries of distress. It is toxic to all vertebrates.[1] It is usually added to grain baits in 0.5%-3.0% concentration, but 25% and 50% concentrates in powdered sugar are available. Recent human exposure has come from its use as an investigational drug in the treatment of multiple sclerosis.[2,3] It is rapidly absorbed by the gut, less effectively across skin. The chief mechanism of toxicity is enhancement of cholinergic transmission in the nervous system through the release of acetylcholine both centrally and peripherally. Due to enhanced transmission at neuromuscular junctions, severe muscle spasms may be a prominent manifestation of toxicity.[2] 4-Aminopyridine is rapidly metabolized and excreted.

No human poisonings have occurred as a result of ordinary use, but the effects of ingestion of about 60 mg each by two adults have been reported. Both experienced immediate abdominal discomfort, nausea and vomiting, weakness, dizziness, and profuse diaphoresis, and one went on to develop a tonic-clonic seizure and required ventilatory support. Acidosis was present in both cases.[1] Dizziness, giddiness, and gait disturbances are common, and seizures may be severe, although recovery with supportive therapy and ventilatory support has been the usual outcome.[1,2,3]

Treatment

1. Skin decontamination. If skin or eye contamination has occurred, thorough washing of the skin or eyes is indicated. See Chapter 2.

2. Gastrointestinal decontamination. If the patient is seen within an hour of ingestion of a significant quantity of this compound, gastrointestinal decontamination should be considered, as outlined in Chapter 2. If treatment is delayed, immediate oral administration of charcoal and sorbitol may represent reasonable management.

3. Seizures may require anticonvulsant medication. See Chapter 2 for dosages.

4. Muscular spasms. Neuromuscular blockade with drugs such as d-tubocuarine, metocurine and pancuronium bromide have been used sucessfully to relieve the muscular spasms that occur with this agent. Such therapy must be provided in an intensive care setting.[1]

5. Dehydration should be treated with intravenous fluids if oral fluids cannot be retained.

CALCIUM CYANAMIDE

This synthetic compound is marketed as granules containing 44% calcium cyanamide, yielding 19.5% nitrogen. It is incorporated into soil to serve as fertilizer, fungicide, and herbicide. In contact with water, hydrogen cyanamide is released. Acidic conditions accelerate this reaction. Hydrogen cyanamide is a solid with considerable vapor pressure. It has toxic properties totally different from those of cyanide, and it does not degrade to cyanide.

Toxicology

Calcium cyanamide is only moderately irritating to skin, but hydrogen cyanamide is severely irritating and caustic to skin and the inhaled gas is strongly irritating to mucous membranes.[4] Dermal and mucosal lesions in the mouth, tongue, and upper esophagus have occurred after exposure. No systemic symptoms from dermal exposure have been reported.[5] Systemic poisonings have followed inhalation of hydrogen cyanamide and ingestion of the salt. Manifestations of poisoning include flushing, headache, vertigo, dyspnea, tachycardia, and hypotension, sometimes progressing to shock.[4] Because cyanamide is an inhibitor of acetaldehyde dehydrogenase, ingestion of alcohol exaggerates the symptoms. (A citrated form of cyanamide has been used in place of Antabuse in alcohol aversion therapy.)

Treatment

1. Skin decontamination. Skin contamination with either the calcium salt or the free form should be removed by washing with soap and water. Flush eyes with copious amounts of clean water. If skin or eye irritation persists, medical attention should be obtained promptly. See Chapter 2.

2. Gastrointestinal decontamination. If large doses have been ingested within an hour of exposure, gastrointestinal decontamination should be considered. If dosage was small or treatment is delayed, oral administration of activated charcoal and sorbitol probably represents reasonable management. See Chapter 2 for doses.

3. Hypotension or Antabuse-type reactions should be treated by placing the patient in the Trendelenburg position, giving intravenous fluids, including plasma or blood, if needed, and, if necessary, vasopressor drugs parenterally.

4. Atropine is not antidotal.

CREOSOTE

Creosote is obtained by distillation of the tar formed by heating wood or coal in the absence of oxygen. It is purified by extraction into oils. Creosote from wood consists mainly of guaiacol (methoxy phenol) and cresol (methyl phenol). Coal-derived creosote contains, in addition, some phenol, pyridine, and pyridinol. Creosote is extensively used as a wood preservative, usually by high-pressure impregnation of lumber. It has also been used as an animal dip and disinfectant. Much of human exposure is in the form of various phenol compounds.

Creosote is irritating to skin, eyes, and mucous membranes. Workers in contact with technical creosote or with treated timbers sometimes develop skin irritation, vesicular or papular eruptions, dermal pigmentation, and occasionally gangrene and skin cancer.[6] Photosensitization has been reported. Eye contamination has resulted in conjunctivitis and keratitis, sometimes resulting in corneal scarring. The constituents of creosote are efficiently absorbed across the skin, but systemic poisonings following dermal absorption have occurred very rarely. Absorption of ingested creosote from the gut occurs promptly, and there may be significant absorption of vapor by the lung. Conjugates of absorbed phenolic constituents are excreted mainly in the urine. Acute toxic effects are similar to those of lysol, but the corrosive nature of creosote is somewhat less because of greater dilution of phenol in the creosote.[7] Irritation of the gastrointestinal tract, toxic encephalopathy, and renal tubular injury are the principal effects. A chronic toxicosis from continuing gastrointestinal absorption (creosote used medicinally) has been described, consisting of gastroenteritis and visual disturbances.

Manifestations of acute systemic poisoning are salivation, vomiting, dyspnea, headache, dizziness, loss of pupillary reflexes, cyanosis, hypothermia, convulsions, and coma. Death is due to multi-organ system failure as patients develop shock, acidosis, respiratory depression, and anuric renal failure.

Confirmation of Poisoning

The presence of phenolic oxidation products imparts a dark, smoky color to the urine.[7] If there is suspicion of poisoning, addition of a few drops of ferric chloride solution to the urine yields a violet or blue color, indicating the presence of phenolic compounds.

Treatment

1. Skin decontamination. Stringent measures should be taken to avoid contamination of skin or eyes and inhalation of vapor. Skin contamination should be promptly washed off with soap and water. Remove eye contamination by washing with copious amounts of water, then obtain specialized medical attention promptly because corneal injury may be severe. See Chapter 2.

2. Gastrointestinal decontamination. If a significant amount of creosote has been ingested and the patient is alert and able to swallow, immediately administer a slurry of activated charcoal by mouth. Further efforts to limit absorption will depend on whether there has been corrosive injury to the esophagus. If pharyngeal redness and swelling are evident, neither induced emesis nor gastric lavage is advisable due to potential re-exposure of the esophagus to the creosote, or perforation of the esophagus from a gastric tube. For further information on gastric decontamination, including charcoal dosing, see Chapter 2.

3. Maintain pulmonary ventilation mechanically with oxygen, if necessary.

4. Blood and urine samples. Draw a blood sample to test for methemoglobinemia, to measure BUN and blood electrolytes, and to check for signs of liver injury (bilirubin, GGT, LDH, ALT, AST, and alkaline phosphatase). Examine the urine for protein and cells, and for "smoky" phenolic excretion products.

5. Intravenous fluids. Give fluids intravenously to correct dehydration and electrolyte disturbances. Include glucose to protect the liver and bicarbonate to relieve metabolic acidosis, as necessary. Monitor fluid balance carefully to signal discontinuation of intravenous fluids if renal failure occurs. Plasma or blood transfusion may be needed to overcome shock.

6. Monitor ECG to detect arrhythmias and/or conduction defects that may appear as manifestations of a toxic myocardiopathy.

7. Convulsions. Anticonvulsants may be needed to control seizures as outlined in Chapter 2.

8. Hemodialysis is not effective in accelerating disposition of phenol (or, presumably, creosote), but hemoperfusion over charcoal probably is effective.[8] This should be considered in severe creosote poisonings.

9. Methemoglobinemia is rarely severe, but intravenous administration of 1% methylene blue may be considered if 25-30% of hemoglobin is converted. Dose is 0.1 mL of 1% solution per kg body weight, given over no less than 10 minutes. Nausea, dizziness, and a transient increase in blood pressure may occur.

ENDOTHALL

As the free acid or as sodium, potassium, or amine salts, endothall is used as a contact herbicide, defoliant, aquatic herbicide, and algacide. It is formulated in aqueous solutions and granules at various strengths.

Toxicology

Endothall is irritating to the skin, eyes, and mucous membranes. It is well absorbed across abraded skin and from the gastrointestinal tract. Recognized systemic toxic mechanisms in mammals are: corrosive effects on the gastrointestinal tract (particularly from high concentrations of the free acid); cardiomyopathy and vascular injury leading to shock; and central nervous system injury, causing convulsions and respiratory depression. A single case has been reported of lethal poisoning in a previously healthy 21-year-old man who died after ingestion of 7-8 grams of endothall. In this patient, hemorrhage and edema were noted in the gastrointestinal tract and lungs.[9] There are no standards for levels, and they are not considered useful in management.

Treatment

1. Skin decontamination. Wash endothall from the skin with soap and water. Flush contamination from the eyes with copious amounts of clean water. Obtain medical attention if irritation of skin or eyes persists. See Chapter 2.

2. Gastrointestinal decontamination. If a large quantity has been ingested, the patient is seen within an hour of exposure, and is fully alert and not convulsing, gastrointestinal decontamination should be considered as outlined in Chapter 2. Lavage is usually contraindicated due to the corrosive nature of this agent.

3. Intubation. If there are indications of corrosive effects in the pharynx, gastric intubation should not be attempted because of the risk of esophageal perforation. Treatment procedures appropriate for ingestions of corrosives (strong acids and alkalis) may be necessary. Referral should be made to a surgeon or gastroenterologist for consideration of endoscopy.

4. Oxygen should be given by mask. If respiratory drive is weak, pulmonary ventilation may have to be supported mechanically.

5. Monitor blood pressure closely. Infusions of plasma, blood, other volume expanders, and pressors may be needed to combat shock.

6. Administer intravenous fluids to correct dehydration, stabilize electrolytes, provide sugar, and support mechanisms for toxicant disposition. Give vasoactive amines very carefully in light of possible myocardiopathy.

7. Convulsions. Seizures may require administration of diazepam and/or other anticonvulsants.

8. Hemodialysis. It is not known whether hemodialysis or hemoperfusion would be effective in removing endothall from the blood. This option should be considered if the patient's condition deteriorates despite supportive care.

METALDEHYDE

Toxicology

Metaldehyde is a four-unit cyclic polymer of acetaldehyde which has long been used to kill slugs and snails, which are attracted to it without the use of bait. Occasional poisonings of animals and children have resulted from ingestion of pellets intended as molluscicide, but tablets designed as a combustible fuel ("meta-fuel") have also been responsible for human poisonings.[10] Another form of exposure is "snow storm tablets," which the user places at the end of a lighted cigarette to create snow. Toxicity occurs through inhalation of metaldehyde fumes.[11] The biochemical mechanism of poisoning is not known. Both acetaldehyde and metaldehyde produced similar effects in dogs; however, acetaldehyde was not detected in the plasma or urine of the metaldehyde-poisoned dogs.[12]

Ingestion of a toxic dose is often followed shortly by nausea and vomiting. The other primary features of toxicity are pyrexia, generalized seizures, and mental status changes, sometimes leading to coma.[10,13] Other signs and symptoms that may occur include hypersalivation, facial flushing, dizziness, tachypnea, and acidosis.[10,11] Pneumonitis has followed inhalational exposure to

metaldehyde.[11] While most cases are dramatic with significant seizures and coma, fatal events are infrequent.[10,13] Poisoned animals show tremors, ataxia, hyperesthesia, salivation, ataxia, and seizures.[12] Autopsy findings in fatal human poisonings indicate severe damage to liver cells and renal tubular epithelium.

Confirmation of Poisoning

Metaldehyde can be measured in the blood and urine, although there are very few reports of levels among poisoned humans. One patient who had severe tonic clonic seizures and was comatose had a metaldehyde level in the serum of 125 mg/L with a half-life of 27 hours. This patient did not have detectable acetaldehyde in the serum.[13]

Treatment

1. Gastrointestinal decontamination. If ingestion occurred within an hour of treatment, consider gastrointestinal decontamination as outlined in Chapter 2. Activated charcoal may well be useful against metaldehyde.

2. Convulsions. If seizures occur, sedative anticonvulsants must be administered. See Chapter 2 for dosage.

3. Supportive treatment. Appropriate supportive treatment including intravenous fluids containing saline and glucose should be given. Sodium bicarbonate may be considered in the event of severe metabolic acidosis. Fluid balance and electrolytes must be monitored carefully to avoid fluid overload if renal failure supervenes.

4. Renal failure. There is no specific antidote for metaldehyde poisoning. Hemodialysis is probably not effective in removing metaldehyde, but must be instituted if renal failure occurs. The effectiveness of hemoperfusion has not been tested.

5. Liver function tests and urine sediment examination should be done to assess liver and kidney injury in poisoned patients.

SODIUM CHLORATE

Sodium chlorate is used in agriculture as a defoliant, nonselective contact herbicide, and semipermanent soil sterilant. Because of its explosive nature, it must be formulated with water-soluble fire retardant material, such as sodium metaborate, soda ash, magnesium chloride, or urea. It is usually applied in water solution.

Toxicology

Sodium chlorate is irritating to skin, eyes, and mucous membranes of the upper respiratory tract.[14] Dermal absorption is slight. Even though gastrointestinal absorption is also inefficient, severe (sometimes fatal) poisoning follows ingestion of a toxic dose, estimated at about 20 grams in the adult human. Excretion is chiefly in the urine. The principal mechanisms of toxicity are hemolysis, methemoglobin formation, cardiac arrhythmia (partly secondary to hyperkalemia), and renal tubular injury.[14,15]

The irritant action on the gut causes nausea, vomiting, and abdominal pain. Once absorbed, hemoglobin is rapidly oxidized to methemoglobin, and intravascular hemolysis occurs.[14] Cyanosis is prominent if methemoglobinemia is severe and may be the only presenting sign.[15] Acute tubular necrosis and hemoglobinuria may result from the hemolysis or direct toxic injury. Plasma and urine are dark brown from the presence of free hemoglobin and methemoglobin.[14,15,16] Release of potassium from red cell destruction results in hyperkalemia which may be severe enough to cause life-threatening arrythmias.[16] The liver and spleen are often enlarged due to uptake of hemolyzed erythrocytes.[15] Hypoxemia may lead to convulsions. Death may be the result of shock, tissue hypoxia, renal failure, hyperkalemia, or disseminated intravascular coagulation (DIC).[14,15,16]

Confirmation of Poisoning

There are no widely available tests specifically for chlorate. Dark brown staining of the plasma and urine indicates the action of a strong oxidizing agent on hemoglobin. See Chapter 2.

Treatment

1. Skin decontamination. Skin contamination should be removed immediately by washing with soap and water. Medical attention should be sought if irritation persists. Flush contamination from eyes with copious amounts of clean water, then obtain specialized medical attention promptly, because irritant action may be severe. See Chapter 2.

2. Gastrointestinal decontamination. If sodium chlorate has been ingested within an hour prior to treatment, consider gastrointestinal decontamination as outlined in Chapter 2.

3. Oxygen. If respiration is depressed, ventilatory support may be necessary.

4. Sodium thiosulfate has been recommended as an antidote against absorbed sodium chlorate. Thiosulfate is thought to inactivate the chlorate ion to form the

less toxic chloride ion. It can be given orally or as an IV infusion over 60-90 minutes. The dose is 2-5 g dissolved in 200 mL of 5% sodium bicarbonate.[14]

5. Monitor blood pressure, fluid balance, blood electrolytes, BUN, methemoglobin, and bilirubin, as well as urine protein, cells and free hemoglobin content, and ECG. Widening of the QRS complex and prolongation of the PR interval indicate hyperkalemic cardiac toxicity.

6. Milk may be helpful in relieving the pain of gastric irritation.

7. Administer intravenous fluids to sustain chlorate excretion. Maintain urine pH in the alkaline range by adding sodium bicarbonate to the infusion fluid. Monitor urine production closely, so that intravenous fluids can be slowed or discontinued if renal failure occurs. Blood transfusion may be needed if hemolysis and methemoglobinemia are severe. Exchange transfusion has been recommended to enhance clearance and treat DIC.[16]

8. Hemodialysis may be life-saving in severe poisoning. It is effective in removing chlorate from the blood, provides a means to control hyperkalemia, and makes possible the control of extracellular fluid volume and composition while renal function remains impaired.

9. Methemoglobinemia. Administration of methylene blue to reverse methemoglobinemia may be considered if as much as 25-30% of hemoglobin is converted. Give intravenously 0.1 mL/kg body weight of a 1% solution over a period of at least 10 minutes. An increase in blood pressure, nausea, and dizziness may occur, but these effects are usually transient. As the use of this agent in chlorate poisoning has not proven beneficial in the past, it is still advisable to proceed to exchange transfusion as stated in #7.

SYNERGISTS:
PIPERONYL BUTOXIDE

Synergists are chemical agents included in pesticide products to enhance the killing power of the active ingredients. The widely-used insecticide synergist, piperonyl butoxide, acts by inhibiting the enzymatic degradation of pyrethrins, rotenone, N-methyl carbamates, and possibly some other insecticides. There is limited dermal absorption on contact. Inherent toxicity in mammals is low. Large absorbed doses may theoretically enhance the toxic hazard of the rapidly metabolized insecticides used today, although inhibition of human drug-metabolizing enzymes by these agents has not actually been demonstrated. Their presence in pesticide products to which humans are exposed does not change

the basic approach to management of poisoning, except that some possibility of enhanced toxicity of the active insecticidal ingredients should be kept in mind.

SOLVENTS AND ADJUVANTS

Liquid materials in which pesticides are dissolved and the solids on which they are adsorbed (sometimes called carriers or vehicles) are selected by producers to achieve stability of the active ingredient, convenience in handling and application, and maximum killing power following application. Often, the particular solvents and adjuvants selected by pesticide manufacturers are responsible for giving their commercial products a competitive edge. For this reason, their inclusion in marketed products is usually proprietary information, not available to the general public except in emergencies. If a poisoning emergency exists, pesticide companies will usually cooperate in supplying physicians with information needed to provide treatment. Some companies will put the inert ingredients on the Material Safety Data Sheet (MSDS). The physician should seek this information to assist in evaluating all possible exposures. A direct request to the producer is the quickest way to secure this information. Physicians may also contact EPA directly for this information (tel: 703-305-7090) if needed for proper management of a case.

Petroleum distillates are the most commonly used solvents for lipophilic pesticides. Most insecticides are lipophilic. The distillates are mixtures of aliphatic and aromatic hydrocarbons and have low boiling points.

Sometimes specific **hydrocarbons,** such as toluene or xylene (strongly odiferous), are added to stabilize the solution of insecticide or make it more emulsifiable. Hydrocarbon-dissolved pesticides are usually diluted for application by adding measured amounts of water to form emulsions. Some chlorinated hydrocarbons may be present in particular technical mixtures. A strong odor lingering after application of a structural pest control spray is often due to the solvent rather than the active ingredient.

Less lipophilic active ingredients are sometimes dissolved in mixtures of alcohols, glycols, ethers, or various chlorinated solvents. It is possible that these enhance the dermal absorbability of some pesticides. Some solvents, such as methanol and isopropanol, may represent a significant toxic hazard if swallowed in sufficient dosage.

Granular formulations utilize various clay materials which adsorb pesticide, retain it in more or less stable form until application, then desorb the material slowly into treated soil. There is some significant desorption when granules are in contact with human skin and very substantial desorption into gastrointestinal secretions if granules are swallowed. The clay materials themselves are not a toxic hazard.

Dusts are infrequently used today. Various forms of talc (silicatecarbonate particles) have been used in the past to adsorb pesticides for application to

foliage. Particle sizes are such that these dusts are usually trapped in the upper respiratory mucous when inhaled. When the mucous is swallowed, the particles desorb pesticide into gastrointestinal secretions. Dust formulations may, therefore, release enough of some pesticides to cause systemic poisonings.

Stickers and spreaders (film extenders) are organic substances added to formulations to disperse pesticide over treated foliage surfaces and enhance adhesion. The availability and persistence of residue on the leaf surfaces is thereby increased. Substances used include proteinaceous materials (milk products, wheat flour, blood albumin, gelatin), oils, gums, resins, clays, polyoxyethylene glycols, terpenes, and other viscid organics. Some also include sulfated alcohols, fatty acid esters, and alkyl and petroleum sulfonates. For persons exposed in the course of formulation or application of pesticides, these adjuvants probably add little or no toxic hazard to that inherent in the active pesticidal ingredients.

Emulsifiers serve to stabilize water-oil emulsions formed when water is added to technical hydrocarbon concentrates. Chemically, they resemble detergents (one part of the molecule lipophilic, the other hydrophilic). Long-chain alkyl sulfonate ethers of polyethylene glycol and polyoxyethylene oleate are exemplary emulsifiers. They have low inherent mammalian toxicity, and their presence probably has little effect on the overall toxicity of formulated products which include them.

Penetrants facilitate the transfer of herbicide from foliage surface to the interior tissues. Some are lipids while others are detergent (surfactant) in nature. Substances used include heavy petroleum oils and distillates, polyol fatty acid esters, polyethoxylated fatty acid esters, aryl alkyl polyoxyethylene glycols, alkyl amine acetate, alkyl aryl sulfonates, polyhydric alcohols, and alkyl phosphates. Some of these are eye and skin irritants, and may account for the irritant effects of particular herbicide formulations whose active ingredients do not have this property.

Safeners are substances added to mixtures of fertilizers with pesticides (commonly herbicides) to limit the formation of undesirable reaction products. Some substances used are alcohol sulfates, sodium alkyl butane diamate, polyesters of sodium thiobutane dioate, and benzene acetonitrile derivatives. Some are moderately irritating to the skin and eyes. Systemic toxicities are generally low.

Anticaking agents are added to granular and dust formulations to facilitate application by preventing cakes and clumps. Among several products used are the sodium salt of mono- and di-methyl naphthalene sulfonate, and diatomaceous earth. Diatomaceous earth has little adverse effect except a drying action on the skin. Methyl naphthalenes are said to be skin irritants and photosensitizers; whether their derivatives have this effect is not known.

Treatment

Petroleum distillates are mineral hydrocarbons which undergo limited absorption across the gut. In general, clinical toxicologists do not recommend induced emesis or gastric lavage in treating ingestions of these materials, because of the serious risk of hydrocarbon pneumonitis if even tiny amounts of the liquid are aspirated into the lung. However, this injunction against emptying the stomach may be set aside when the petroleum distillate is a vehicle for toxic pesticides in significant concentration. In such cases, if the patient is seen within one hour of exposure, gastrointestinal decontamination should be considered.

Rapid respiration, cyanosis, tachycardia, and low-grade fever are the usual indications of frank hydrocarbon pneumonitis. Patients with presumed hydrocarbon pneumonitis, who are symptomatic, should usually be hospitalized, preferably in an intensive care setting. If the patient has pulmonary symptoms, a chest x-ray should be taken to detect or confirm signs of pneumonitis. In addition, the urine should be examined for protein, sugar, acetone, casts, and cells, and an ECG should be examined for arrhythmias and conduction defects. Mechanically assisted pulmonary ventilation with 100% oxygen may be required. Hydrocarbon pneumonitis is sometimes fatal, and survivors may require several weeks for full recovery. In milder cases, clinical improvement usually occurs within several days, although radiographic findings will remain abnormal for longer periods.[17]

The presence of chlorinated solvents in some formulations may add significantly to the toxic hazard, particularly if the product is ingested. Certain adjuvants are irritants to skin, eyes, and mucous membranes, and may account for the irritant properties of some products whose active ingredients do not have this effect. With these exceptions, however, the presence of adjuvants in most finished pesticide products probably does not enhance or reduce systemic mammalian toxicity to any great extent.

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