R7DHRE Hazardous Materials Guideline: Phosphine

Region VII Disaster Health Response Ecosystem

REGION VII DISASTER HEALTH RESPONSE ECOSYSTEM (R7DHRE)

CHEMICAL SPECIALTY TEAM

Call Your Poison Center for Immediate Assistance: 1-800-222-1222

Hazardous Materials Guideline: Phosphine

This document is intended as a supplement for discussion with your local poison center or toxicologist.

1.0 BACKGROUND

1.1 Description: Phosphine is a colorless, extremely flammable, explosive, toxic gas with an odor of decaying fish or garlic. It can ignite spontaneously on contact with air. Phosphine is heavier than air and may also cause simple asphyxiation in enclosed, poorly ventilated, or low-lying areas. Aluminum phosphide and zinc phosphide are solid pellets which are used as a grain fumigant and a rodenticide, respectively. When phosphides are ingested or exposed to moisture, they release phosphine gas. Phosphine gas can be shipped in cylinders as a liquefied compressed gas; contact with liquid phosphine may result in frostbite injury.

1.1.1 Phosphine is a respiratory irritant and systemic poison. It is NOT the same as phosgene (a respiratory irritant) or phosgene oxime (a vesicant).

1.2 Mechanism of Injury: Phosphine itself is a severe respiratory tract irritant. Phosphine is also a chemical asphyxiant and as such it is a highly toxic systemic poison. It primarily affects the mitochondria of heart and lungs, leading to pulmonary edema, CNS depression, cardiovascular collapse (severe peripheral vascular dilatation with cardiac failure) and cardiac arrest.

1.2.1 Burning of phosphine produces phosphorus pentoxide, which dissolves in the water of the respiratory tract to produce phosphoric acid, another severe respiratory tract irritant.

1.3 Routes of Exposure: Inhalation, Ingestion, Dermal.

2.0 PROVIDER SAFETY

2.1 Personal Protective Equipment (PPE) – Decontamination Team: Personnel decontaminating patients exposed to solid phosphides must wear full-body chemical-resistant clothing and respiratory protection. Respiratory protection may consist of either:

2.1.1 A positive pressure air or oxygen source, such as an air-line respirator or a Self-Contained Breathing Apparatus (SCBA) or

2.1.2 A filtered air respirator (including Powered Air Purifying Respirators (PAPRs)) with filters capable of adsorbing phosphine.

2.1.3 A positive pressure air or oxygen source is preferred if there is doubt as to the identity of the chemical in question or if there may be exposure to a level of phosphine which would overwhelm the filter.

2.2 Personal Protective Equipment (PPE) – Treatment Team: Personnel treating patients who have been adequately decontaminated need no additional PPE other than **universal precautions** since there is no serious risk of secondary contamination.

2.3 Patient Decontamination:

2.3.1 Persons exposed to only **phosphine gas** and have **no skin irritation, no eye irritation, dry skin, AND dry clothes generally do not need decontamination**. These patients do not pose a significant risk of secondary contamination.

2.3.1.1 The vomitus from persons who have ingested aluminum or zinc phosphide is hazardous because it may off-gas phosphine or contaminate those coming in contact with the vomit. Prepare treatment areas for rapid clean up in case the patient vomits.

2.3.1.2 Those persons contaminated with **solid phosphides do pose a risk of secondary contamination** if not properly decontaminated. Metallic (aluminum, zinc) phosphides on clothes, skin, or hair can react with water or moisture to generate phosphine gas.

2.3.2 Brush phosphide powder from the skin, hair, and clothes of victims.

2.3.3 Remove contact lenses if it can be done without additional trauma to the eye. **Irrigate eyes for a minimum of 15 minutes**. Continue irrigation until eye pH is neutral (7 to 8).

2.3.4 Remove ALL clothing and jewelry. Double bag clothing and jewelry to prevent off-gassing.

2.3.5 Decontamination is best **accomplished by irrigation with copious amounts of water**. Wash skin and hair with plain water for a minimum of 5 minutes and then wash twice with soap & water after washing with plain water. Washing with water alone (for a longer period of time) is acceptable if soap is not available. **Neutralization is NOT recommended**. Skin pH can be checked to assure that all of the phosphine has been removed.

2.3.5.1 **REMEMBER**: Phosphide + Water = Phosphine

2.3.6 Watch for hypothermia (1) in children and the elderly, (2) when decontamination is done with un-heated water, or (3) during cold weather.

3.0 SIGNS & SYMPTOMS

3.1 Severity of symptoms will depend upon the concentration of the phosphine to which the person is exposed and the duration of exposure. Most deaths occur within the first 12 to 24 hours after exposure and are cardiovascular in origin. Deaths after 24 hours are usually due to liver or renal failure. Some systemic symptoms may be delayed for up to 72 hours after exposure.

3.2 Inhalation: Chest tightness, cough, dyspnea, retrosternal discomfort, and tachypnea. Severe exposure can cause pulmonary edema, pneumonitis and ARDS, which may have a delayed onset of 72 hours or more after exposure.

3.3 Dermal: Phosphides may be absorbed dermally, especially through broken skin, and can cause systemic toxicity by this route. Contact with liquefied or compressed phosphine gas may cause frostbite.

3.4 Ocular: Irritation, burning, tearing and conjunctivitis. The full extent of eye damage may not be fully evident for several days.

3.5 Ingestion: Onset of symptoms after the ingestion of a phosphide is usually within several minutes. Phosphide tablets can act as a direct corrosive to the stomach and GI tract. Symptoms include nausea, vomiting, abdominal pain, diarrhea, and ileus.

3.6 Systemic: Systemic effects are more likely to occur after inhalation or ingestion but may also occur after dermal exposure.

3.6.1 Cardiovascular: Hypotension, decreased cardiac contractility, severe peripheral vasodilatation, tachycardia, arrhythmias, cyanosis, and cardiac arrest. Abnormal myocardial enzymes may be seen, and significant hypomagnesemia and hypermagnesemia have been associated with massive focal myocardial damage. EKG changes, including ST-T changes and conduction abnormalities, can occur. If the patient survives the initial 24 hours, the ECG typically returns to normal, indicating that heart damage is reversible.

3.6.2 Neurologic: Headache, restlessness, irritability, drowsiness, dizziness, diplopia, ataxia, vertigo, loss of feeling, tremors, diminished reflexes, seizures, and coma.

3.7 Renal: Oliguria and anuria from poor renal perfusion. Acute renal failure may occur. Metabolic acidosis is likely caused by poor perfusion and cellular anoxia.

3.8 Hepatic: Liver injury (elevated serum transaminases, jaundice, hepatosplenomegaly) usually appears 48-72 hours post-exposure. Centrilobular necrosis has occurred in fatal cases.

4.0 DIAGNOSTICS

4.1 Phosphine poisoning is a clinical diagnosis and there is no specific diagnostic testing. Any diagnostic evaluation should be based on sign and symptoms.

4.2 Basic labs (complete metabolic panel, lactate, troponin) for any symptomatic patient.

4.3 Consider an EKG to assess for signs of cardiac ischemia.

4.4 Consider a chest x-ray in patients with persistent symptoms and hypoxia.

5.0 TREATMENT

5.1 General: Follow standard Basic and Advanced Life Support Guidelines. There is no specific antidote for phosphine poisoning. Hypotension may be poorly responsive to vasopressors. Symptomatic patients should be observed for at least 72 hours.

5.2 Inhalation: Maintain the patient's airway, with endotracheal intubation or cricothyroidotomy if necessary. Endotracheal intubation should be performed only under direct visualization because of edema and potential damage to the oropharynx. Support oxygenation and ventilation as necessary. Use standard treatments for pulmonary edema (diuretics, PEEP, etc.) and bronchospasm (inhaled bronchodilators; consider corticosteroids).

5.3 Dermal: **Treatment is the same as that for thermal burns**. If frostbite is present, rewarm the affected area in the same manner as for environmentally induced frostbite.

5.4 Ocular: **Irrigate eyes to a neutral pH**. Perform a thorough eye exam: test visual acuity, and perform fluorescein and slit lamp examinations. Ophthalmology consultation may be necessary. Immediately consult an ophthalmologist for patients who have corneal injuries.

5.5 Ingestion: Do **NOT** give induce emesis. Consider administering activated charcoal, 1 gm/kg orally, **ONLY** to patients who are conscious, able to swallow, and are able to protect their airway. Activated charcoal should not be given if the patient will need endoscopy.

5.5.1 Gastric lavage may be considered, but the addition of water to metal phosphides can create an exothermic reaction. If gastric lavage is being considered, use of a potassium permanganate solution (1:10,000) has been recommended; the permanganate oxidizes phosphine in the stomach to phosphate, thus reducing the available phosphine.

Disclaimer: This guideline is intended to be an informational reference only and should not be used as a substitute for consultation with a poison center or toxicologist, and/or the clinical judgement of the bedside team.

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