Florida Poison Information Center/Jacksonville
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University of Florida Health Science Center
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Sulfur Mustard

History

Sulfur mustard was first synthesized in the early 1800s and first used on the battlefield during World War I by Germany. It produced most of the chemical casualties, although less than 5% of those who reached medical treatment facilities died. Today, mustard is still considered a major threat agent of former Warsaw Pact countries and third world countries. The United States manufactured mustard during both World War I and II and maintains a stockpile that is currently undergoing destruction.

Mechanism of Action

Following absorption into the body, mustard rapidly cyclizes in extracellular water. The resulting cyclic compound quickly binds to intra and extracellular enzymes, proteins, and other substances. The exact mechanism by which mustard produces tissue injury is unknown. One hypothesis is that damage results from DNA alkylation and crosslinking in rapidly dividing cells such as basal keratinocytes, mucosal epithelium, and bone marrow precursor cells. This leads to inflammation and cellular death. Protease digestion of anchoring filaments in the epidermal-dermal junction results in formation of blisters. Mild cholinergic effects may be responsible for effects such as early gastrointestinal symptoms and miosis. Once mustard reacts with body tissues, it is no longer an intact molecule. Therefore one cannot become exposed from contact with body fluids or tissues.

Properties

Mustard is an oily liquid ranging from light brown to yellow in color and has an odor similar to garlic, onion, or mustard. Under temperate conditions mustard evaporates slowly posing more of a liquid hazard, but with increasing temperature its vapor hazard increases and is considered a definite vapor hazard at temperatures of 100ºF or greater. Mustard freezes at 57ºF and is often mixed with substances with a lower freezing point, e.g. Lewisite, so that the mixture will remain liquid at lower temperatures. Mustard may be dispersed by spraying or by the explosive blast from a shell or bomb.

Symptoms

Skin- Erythema may appear within 2 to 24 hours following vapor exposure. Time of onset is dependent on concentration, ambient temperature and humidity, and skin site exposed. More sensitive sites are warm moist locations with thin skin. Small vesicles can develop within erythematous areas that may later coalesce to form bullae. Fluid within the blister does not contain mustard and is not a vesicant. Exposure from higher doses, such as those from liquid contact, may result in lesions with a central area of coagulation necrosis.

Pulmonary- The primary lesion is necrosis of the laryngeal and tracheobronchial mucosa with later damage to the musculature of the airways. The earliest effects may be irritation/burning of the nares, epistaxis, sinus pain, and irritation or soreness of the pharynx. Larger concentrations

and prolonged exposure leads to progressive injury and involvement of the lower airway. Symptoms may progress to severe cough, dyspnea, and on rare occasion hemorrhagic pulmonary edema. The primary cause of death in mustard poisoning is respiratory distress and failure, whether it is from 1) airway edema 2) mechanical obstruction from pseudomembrane formation or tissue necrosis or 3) bacterial pneumonia.

Ocular- Low-dose vapor exposure may result only in irritation. Patients may present with miosis secondary to cholinergic activity. More severe conjunctivitis, photophobia, blepharospasm, pain, and corneal damage are seen with larger doses. Severe effects may result in eventual scarring between the iris and lens restricting pupillary movement and predisposing the victim to glaucoma. More severe damage is caused by liquid mustard from airborne droplets.

Gastrointestinal- The mucosa of the GI tract is very susceptible to mustard although reports of severe GI effects from exposure are infrequent. Nausea is the primary symptom although diarrhea (rarely bloody) and vomiting may occur after high-dose exposure and implies a poor prognosis.

CNS- Animal studies suggest that mustards (particularly nitrogen mustards) are convulsants although the CNS effects are poorly defined in humans.

Medical Management

1. Remove all contaminated clothing and handle as hazardous waste. Wash any remaining material off of the body with large amounts of soap and water. Management at this point becomes primarily supportive.
2. Erythema may be treated with calamine or other soothing lotion or cream to reduce burning and itching. Denuded areas should be thoroughly cleaned and treated with a topical antibiotic. Fluid loss is not of the magnitude seen with thermal burns although fluid and electrolytes should be carefully monitored.
3. Eyes should be thoroughly irrigated followed by regular application of a mydriatic (helps to prevent future synechiae formation) and a topical antibiotic.
4. Mild upper airway symptoms may respond to cough suppressants. Symptoms suggestive of bronchitis may appear 12 to 24 hours following exposure and are usually secondary to a sterile process or pneumonitis. However increasing fever, dyspnea, and pulmonary infiltrate on x-ray may indicate infection and should be treated appropriately with antibiotics. Intubation may be necessary in those with severe pulmonary effects.

Bibliography

US Army Medical Research Institute of Chemical Defense: Medical Management of Chemical Casualties Handbook. 1995

Treatment of Chemical Agent Casualties and Conventional Military Chemical Injuries. Departments of the Army, Navy, Air Force, and Commandant, Marine Corps.

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