Nursing Care of Acute Sulfur Mustard Poisoning

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Introduction

Mustard poisoning although rare, has serious consequences. During the last decades, use of mustard gas, a debilitating chemical weapon, left numerous casualties behind. Recent threat of terrorist attacks on the world made us more cautious about teaching those nursing techniques that are not readily available elsewhere. Herein, we present the nursing care of patients with acute sulfur mustard (SM) poisoning.

There are two types of mustard—SM and nitrogen mustard. The former, also known as the “blister agent,” has been used as a chemical weapon. Nitrogen mustard, however, has no weaponry use and has been used in chemotherapy of cancer.

SM is a fat soluble yellow-brownish substance. As an alkylating agent, SM damages nucleic acids and proteins, impairs cell homeostasis and eventually causes cell death. It rapidly reacts with ocular, respiratory and cutaneous tissues, as well as bone marrow and the mucosal cells of the gastrointestinal tract, resulting in several devastating long-term effects on human health.

SM can be absorbed through the respiratory tract, skin, and cornea. It can also be absorbed through the gastrointestinal tract after consumption of contaminated food.1

The acute toxic effects of SM usually follow a symptom-free period. This period varies from a few minutes to several hours, depending on the exposure dose, route of toxicity, ambient temperature and some individual characteristics like his/her sensitivity to SM.1,2

Toxic Effects of Sulfur Mustard on Body Organs

Eyes
Eyes are the most sensitive organ to SM. Just a few hours after exposure, corneal epithelium develops blisters and sloughing. Visual acuity drops significantly. With higher doses of SM, cornea develops ulcer which rarely results in permanent blindness.1

Respiratory Tract
After eyes, the most susceptible organ to SM is the respiratory tract. The first (sometimes the only) symptom after exposure is pain and irritation in the nasal cavity and sinuses; this usually happens 4–6 hours after exposure. The victim may develop epistaxis and non-productive cough. In those who exposed to a high concentration of SM, the epithelial inflammation and necrosis may produce a clinical picture resembling the pseudomembrane of diphtheria. This membrane can be developed anywhere in the respiratory tract and cause obstruction. Cough and hoarseness may last for six weeks.1,2

Skin
The typical skin lesions developed after an exposure to SM are erythema followed by blisters. Erythema usually develops 2–24
hours after exposure and is associated with severe pruritus. Typically, within 18 hours of exposure, patient develops vesicular lesions which gradually join to make bullae containing a large amount of yellow fluids, the number of which increases over 48 hours of exposure (Fig. 1). The bullae are not painful, themselves.

Gastrointestinal tract
The most common gastrointestinal symptoms after SM exposure are nausea, vomiting, anorexia, abdominal pain and diarrhea. Presence of nausea and vomiting occurring within first 24 hours of exposure is just a reflex and does not reflect any damage to the epithelial lining.

First aid measures
The victim should take away as soon as possible from the contaminated area. All cloths should be taken off and destroyed. The skin should be washed with copious amounts of water and a neutral soap (pH near 7). The injured site may also be washed with olive oil followed by soap and water. In case of contamination with liquid mustard, eyes should instantly be washed with large amounts of water, normal saline or Ringer’s solution. Those who have mild symptoms should be observed for at least 24 hours. Severely injured patients should be hospitalized.

Nursing Care

Eyes
Since SM reacts rapidly with corneal tissue and causes serious irreversible damage, eyes should be washed as soon as possible with large amounts of water, even in symptom-free patients. Washing after 10–15 minutes of exposure is of little benefit. Although numerous ophthalmic drops like pure water, normal saline, sodium bicarbonate 1.5%, saturate solution of sodium or magnesium sulfate, and boric acid have been proposed to alleviate the inflammation, we found that normal saline is the eye drop of choice. Vaseline can be used to prevent palpebral adhesion. However, its use should be delayed until no SM exists in the environment as applying this oily compound may result in the accumulation and concentration of the residual SM left in the environment. Mydriatics and cycloplegics should be administered to treat the pain and prevent posterior synechiae. Use of topical ophthalmic analgesic drops should be avoided unless it is really necessary (e.g., ophthalmic examinations) since they are mostly poisonous. Use of topical steroids should also be avoided. No eye pad should be used as it may increase the temperature of the eye which accelerates the noxious effects of SM. Use of sunglasses is encouraged. The mainstay of the therapy is repeated irrigation of the affected eye with copious amounts of water or other appropriate solutions. Special attention should be made to assess the psychological status of patient.

Respiratory tract
To prevent pulmonary edema, beclometh-
asone inhaler should be administered as soon as possible. The patient should receive cold pneumatized air and chest physiotherapy to expel excessive mucus. Patient should place in a semi-sitting position for ease of mucus discharge. Appropriate antibiotic therapy should also be started on a regular basis to prevent secondary lung infection. Use bronchodilators in those with sensitive airway disease. In case of severe damage to lungs, patient should be admitted to intensive care unit and if necessary, undergo mechanical ventilation. Patients should receive oxygen and the level of oxygenation be monitored with pulse-oximetry.1,2

Skin
The affected skin should be washed with 0.2%–0.3% chloramine-T solution for at least three times a day and dressed with silver sulfadiazine 1% cream to prevent secondary infections. Calamine lotion and topical steroid solutions can be used to alleviate itching and dermal irritation. Skin debridement should be done if necessary. Systemic antibiotic therapy should be started. Acetaminophen, morphine, systemic antihistaminics and tranquilizers may be used to control the severe pain and pruritus. Bullae ≤2 cm in diameter should be left intact. However, if they are punctured spontaneously, debridement is necessary. The fluid in bullae >2 cm in diameter should be sucked by a syringe and they should be debrided, washed with normal saline and dressed with silver sulfadiazine cream. The room temperature should be temperate and patients should be on high calorie, high protein diet. Here, the mainstay of therapy aims at preventing secondary wound infection.1,2

Bone-marrow depression
Patients with bone-marrow suppression, leukopenia and aplastic anemia should be isolated and received packed red cell and platelet. Complete blood cell count and platelet count should be requested regularly. Nursing care is similar to that of patients with blood dyscrasia.4

Delayed Complications
Toxic effects of SM in severely intoxicated patients persist for their entire life. They need life-long medical and nursing care provided according to the protocols developed by the research group of Medical Toxicology Research Center (MTRC), Imam Reza Hospital, Mashhad University of Medical Sciences.5-8

References
3. Helm UK, Balali-Mood M. Cutaneous lesions

TAKE-HOME MESSAGE
- Sulfur mustard affects many systems including, eyes, respiratory tract, gastrointestinal tract, hematopoietic system and the skin.
- Nursing care of patients with sulfur mustard poisoning should be started from the time of exposure and continue life-long.
- Bullae ≤2 cm in diameter should be left intact; the fluid of larger ones should be sucked by a syringe.
produced by sulfur mustard. The First International Medical Congress on Chemical Warfare Agents in Iran. Mashhad, Iran: Mashhad University of Medical Sciences; June 13-16, 1988: No. 90.


In our region, many boys and girls are shepherds and at risk of developing brucellosis and/or hydatid disease. (See pages 62 and 88)