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CUTANEOUS AND OCULAR LATE COMPLICATIONS OF SULFUR MUSTARD IN IRANIAN VETERANS

Majid Shohrati, Masoud Davoudi, and Mostafa Ghanei
Research Center of Chemical Injuries, Baqiyatallah University of Medical Sciences, Tehran, Iran

Mohammadreza Peyman
Young Researchers Club, Tehran, Iran

Alireza Peyman
Isfahan University of Medical Sciences and Health Services

Although sulfur mustard (SM) has been used as a chemical warfare agent since the early twentieth century, it has reemerged in the past decade as a major threat around the world. This agent injured over 100,000 Iranians and one-third is suffering from late effects until today. Mustard affects many organs such as the skin, eyes, and lungs, as well as the gastrointestinal, endocrine, and hematopoietic system.

In this study we focused on review of the late Cutaneous and ocular complications caused by exposure to SM.

All studies regarding long-term ocular and cutaneous effects, which have been done on Iranian population, were collected from domestic and international sources. Pruritus is the most common complain and a malignant change is the most important lesion, which has to be considered. Also this agent is causes of chronic and delayed destructive lesions in the ocular surface and cornea, leading to progressive visual deterioration and ocular irritation.

Keywords: Cutaneous; Late complication; Ocular; Sulfur mustard

INTRODUCTION

Chemical warfare agents are potentially accessible because they are easily and inexpensively produced. This means that they are ideal to use by terrorists and in military operations against civilian populations and troops (1).

The North Atlantic Treaty Organization's definition of a chemical agent is "a chemical substance which is intended to use in military operations to kill, seriously injure or incapacitate people because of its physiological effects" (2).

During the studies of interactions between olefins and sulphur halogen compounds mustard gas (bis (2-chloroethyl) sulphide)) was first synthesized in 1822 by Despretz and in 1860 by Niemann and Guthrie (3,4).

Address correspondence to Majid Shohrati, PhD, Baqiyatallah University of Medical Sciences, P.O. Box: 19945-546, Tehran, Iran. E-mail: shohratimajid@yahoo.com

Table 1 Physico-chemical properties of sulphur mustard (5)

		Sulphur mustard
Appearance	–	Yellow, oily liquid
Odour	Distilled	Nearly no odour
–	Technical grade	Garlic, mustard
Molecular weight	–	159.08
Density	–	1.27 (specific gravity)
Solubility	–	Very hydrophobic
Freezing point	–	14.45°C
Boiling point	–	215–217°C
Volatility (mg/m^3, 20°C)	–	610
Persistence	–	High (days–weeks) stable during rain because of slow hydrolysis

Pure Sulphur Mustard (SM) is a transparent, colorless liquid, which is nearly odorless. Its freezing point is 14.4°C and its boiling point is between 215°C and 217°C (760 mmHg). The physico-chemical properties are given in Table 1 (5).

SM is the vesicant with the highest military significance. Its last military use was in the Iran—Iraq war (6). This agent injured over 100,000 Iranians and one-third is suffering from late effects until today (7). Firstly it was widely used during World War I (8). There have been reports of chemical attacks in which sulfur mustard might have been used (a) on Iranian soldiers and civilians during the Gulf War in 1984 and 1985 and (b) in an Iraqi chemical attack on the Iranian-occupied village of Halbja in 1988, resulted in many civilian casualties. Heavy use of chemical warfare in Afghanistan by the Soviet military is a recent innovation in military tactics that has been highly successful and may ensure further use of chemical agents in future military conflicts and terrorist attacks as a profitable adjunct to conventional military arms (9). It also was used in chemical warfare in Ethiopia in 1936 (10).

Mustard affects many organs such as the skin, eyes, and lungs, as well as the gastrointestinal, endocrine, and hematopoietic system (11–13).

Incidence of lungs, eyes, and skin lesions as late complications in 34000 Iranians with wartime exposure to mustard agent is mentioned as 42.5%, 39.3%, and 24.5%, respectively (14).

In this study we focused on review of the late Cutaneous and ocular complications caused by exposure to SM.

MATERIALS AND METHODS

All studies regarding long-term ocular and cutaneous effects, which have been done on Iranian population, were collected from textbooks, domestic, and international articles in medical journals and online databases. To access related studies in Electronic databases, key words such as mustard gas, chemical warfare, sulfur mustard, Lost, yellow cross, dichlorediethyle sulfide, HD, and Iran were used.

MECHANISM OF ACTION

After absorption, SM undergoes intramolecular cyclization to form an ethylene episulphonium ion intermediate (15). Conversion to this hyperactive compound is facilitated in aqueous solution (16), which accounts for the sensitivity of mucosal tissues to its reaction. The cyclic intermediate reacts avidly with and alkylates a wide variety of electron-rich biological molecules such as the sulphydryl (−SH) and amino ($-NH_2$) groups of proteins and nucleic acids (5,17).

Among difunctional alkylating agents, mustard with two chloroethyl groups, appears to exert a more potent cytotoxic activity than monofunctional ones do such as the hemisulphur mustard, 2-chloroethyl-2-hydroxyethylsulphide, which has merely one chloroethyl group (18). The monofunctional mustards, have one alkylating site and therefore can attack and break the DNA at specific nucleotides. The major alkylating site of nucleic acids in mammalian origin is the nitrogen residue of guanine (17). Bifunctional mustards, however, attack an additional guanine residue to achieve inter- and intrastrand DNA crosslinking (19,20). The results are manifested in chromatid aberrations and inhibition of DNA, RNA, and protein synthesis. Although SM reacts with RNA, proteins, and phospholipids, it is widely accepted that it is DNA alkylation, which plays an important role in delayed healing (21).

The interstrand DNA cross-link produced by bifunctional mustard compounds probably induces the lesion that is lethal at the lowest frequency of occurrence and at the lowest concentration of the agent. However, cell death from this lesion is delayed until the cell replicates its DNA or undergoes division. At higher cellular exposure, mechanisms other than DNA cross-linking become important and induce more rapid cell death. The acute damage experienced with SM to the cornea, mucous membranes, and skin; is probably generated by one or more of these other mechanisms.

One mechanism that may be involved in acute damage is nicotinamide adenine dinucleotide (NAD) depletion. Single-strand breaks within DNA trigger activation of DNA repair enzymes, particularly poly-(adenosine diphosphateribose) polymerase (PADPRP). Excessive PADPRP activity depletes cellular NAD^+, and this, in turn, inhibits glycolysis, leads to release of tissue proteases, and ultimately results in cell necrosis (22–24). Proteases released from epidermal cells are assumed to cause disruption of dermal–epidermal attachments and subsequent blister formation (5).

Other potential mechanisms of cell death are related to rapid inactivation of sulphydryl-containing proteins and peptides such as glutathione. These sulphydryl compounds are critical in maintaining the appropriate oxidation–reduction state of cellular components. Glutathione is also thought to be critical in reducing reactive oxygen species in the cell and preventing peroxidation and loss of membrane integrity (23,24).

Also after exposure, cells try to repair the damaged DNA and recover or die. Keratinocyte cell death can be either by apoptosis, necrosis or terminal differentiation (25).

The most investigated pharmacological compounds for treating sulphur mustard injuries are intracellular scavengers (e.g., *N*-acetyl-cysteine, amifostine), cell cycle inhibitors (e.g., mimosine), PARP inhibitors (niacinamide), calcium modulators (e.g., BAPTA), protease inhibitors (e.g., sulfonyl fluorides, Ilomastat), and anti-inflammatory compounds (e.g., indomethacin) (26). But until now, a specific antidotal treatment is missing.

CUTANEOUS COMPLICATIONS

In terms of cutaneous injury, vesicants—mainly SM is the most significant chemical warfare agent (1). It is a powerful blistering agent; a 0.1 mL drop of pure SM contains 20,000 times the dose required to blister skin (27).

Penetration rates of liquid SM measured in vitro (71–294 $\mu g/cm^2/h$, human skin) and in vivo in human volunteers (60–240 $\mu g/cm^2/h$) were found to be similar (28,29). Twenty micrograms per centimeter square of liquid sulphur mustard on skin are sufficient to produce blisters (30), but only 4 $\mu g/cm^{-2}$ of vapor sulphur mustard is needed for this effect (29).

Overview of all performed studies reveals that pruritus is the most common and important complain of SM exposed patient suffering from chronic skin lesions (70–90% of patients), followed by burning (15–50%), pain (3–10%) and redness (6–10%) (30–34). Xerosis (dry skin) is the main and most frequent skin finding in physical examination (25–55%) (31,32). Hyper pigmentation (20–50%) and hypo pigmentation (2–15%) are other common skin signs, followed by mustard scar (2–13%) (33,34). Mustard scar is actually hallmark of SM exposure in skin lesions, which can be detected in different shapes such as atrophic changes, hypertrophic scar and keloid formation (35). Vascular changes, including telangiectasia and cherry angioma, are other observed findings (35). Overall, according to reports of Iranian studies, there are some specific signs in skin lesions that can be divided in the following groups: 1) salt and pepper pigmentation changes; 2) pigmentation changes along with vascular changes like telangiectasia, cherry angioma; and 3) In addition to above-mentioned signs hypo or hyper trophic lesions can also be observed. These lesions can be detected mainly on face and around eyes, neck, external genitalia, axillae, and antecubital fossae (30–35).

Clinically the most frequently involved areas were genitalia, face, and axilla. As it is mentioned above the most common cutaneous findings were erosions, erythema, and hyperpigmentation. The histopathologic changes of skin induced by mustard gas, includes four distinct patterns: 1) Interface dermatitis, vacuolar type and lichenoid type, 2) Spongiotic dermatitis and bullous dermatitis (with or without acantholysis), 3) Pigmentary disorder pattern, increase of epidermal melanization, and 4) Alteration of dermis/hypodermis, sclerodermoid pattern, vasculopathy, and appendageal inflammatory response. Despite some specific characteristics related to sulfur mustard effect, these findings were compatible with histopathological changes of the chemical burns (36).

According to skin disorders reported in Iranian studies, there are disorders that their presence can be associated to SM exposure. Eczema, seborrheic dermatitis, cherry angioma, and urticaria account for most common skin diagnosed disorders in this group (32–35). On the other hand, there are other disorders (such as Androgenic alopecia, Psoriasis, erythematic discoid lupus, lichen planus, Tinea versicolor, and vitiligo) that need to ascertain their association with SM exposure requires to be studied more in future (37,38).

Despite the fact that SM is a carcinogenic agent and there are some reports regarding the high prevalence of skin cancers in patients with history of SM exposure, overview, and appraisal of present studies do not support high risk of cancer occurrence in Iranian and First World War victims who had short-term exposure

to this agent (39). A part of this can be pertained to methodological errors (ignorance of consideration of confounding factors) in these studies (31). What is more, the duration of 17–22 years after exposure is not long enough to conclude the association of skin cancers (such as BCC, SCC, and Melanoma) with SM exposure (40).

However, because of chronic exposure to SM and consequently impaired repair mechanism of DNA in sulfur mustard factory workers, high risk of skin cancer can be associated to SM exposure (41). Overall, the longer following up of these patients can be drawn by the conclusion regarding the association of acute SM exposure and high prevalence of skin cancer.

Treatment in the chronic phase is mainly symptomatic. Systemic antihistamines and local emollients can help to reduce itching and improve skin dryness. Frequent baths should be discouraged in these patients. Sunscreen lotions or creams can also be applied for hyper-pigmented lesions. Contractures rarely occur with chemical burns caused by SM.

OPHTHALMIC COMPLICATIONS

The most common ocular effects were conjunctivitis and photophobia. Patients with significant corneal involvement are at risk for corneal ulceration and rarely for anterior chamber scarring and neovascularization, any of which would result in prolonged disability. To put it in a nutshell, the eye is the most sensitive organ to SM vapor. Ocular injuries generally heal completely. In severe cases, blindness may occur. The need for corneal transplantation is rare (41).

SM reacts rapidly with ocular tissues, and after a latent period of a few hours the patient starts suffering from severe eye pain, photophobia, excessive lacrimation, and blindness. The injury, which is restricted to the anterior segment of the eye, may cause long-lasting incapacity in large numbers of casualties. Approximately 0.5% of the severely wounded victims may develop late complications, which require prolonged ophthalmologic observation and therapy. In light of the ever-present threat of mustard chemical warfare against military and civilians, physicians worldwide should be aware of its grave effects and know how to care for its victims (42).

In report of 48 patients with chronic and delayed-onset mustard gas keratitis, Javadi et al. concludes that mustard gas causes chronic and delayed destructive lesions in the ocular surface and cornea, leading to progressive visual deterioration and ocular irritation. Based on the clinical appearance of the lesions and the histopathologic findings, an immune-mediated component seems possible (43). In a study, the symptoms in 40 SM exposed patients were blurred vision (50%), itching (42.5%), burning sensation (37.5%), photophobia (30%), tearing (27.5%), reading difficulties (10%), red eye (10%), eye pain (2.5%), and foreign body sensation (2.5%). Objective and subjective refraction and Snellen acuity charts were used to determine the best corrected visual acuity at distance. The best-corrected visual acuity was equal or better than 20/25 in 28 patients, between 20/40 and 20/25 in eight patients, and worse than 20/40 in only three patients. Schirmer test for dryness of the eyes revealed mild dryness (5–10 mm) in eight patients and severe dryness (<5 mm) in four patients. Slit lamp examination of the conjunctiva was normal in eight patients. In the other patients, pinguecula (62.5%), ptyrigium (17.5%), and chronic conjunctivitis (17.5%) were diagnosed. Abnormal findings in the limbus were recorded as peri-limbal

hyperpigmentation (17.5%), vascular tortuosity (15%), and limbal ischemia (12.5%). Slit lamp examination of the cornea was normal in 26 patients. In the other patients, subepithelial opacity (15%), corneal thinning (15%), severe opacity (10%), micro/macro pannus (7.5% each), corneal vascularization (7.5%), and corneal epithelial defect (5%) were recorded. Intraoccular pressure and fundoscopy were normal in all patients. Severity grading was determined as 14 (35%) patients in grade 1, 15 (37.5%) in grade 2, 5 (12.5%) in grade 3, and 6 (15%) patients in grade 4 (44).

To date there has been no definite treatment for the delayed keratitis caused by SM. However, artificial tears (45), therapeutic contact lenses (46–49), local/systemic corticosteroids and other immunosuppressive drugs such as azathioprin may be used according to the severity of keratitis (45,46). Corneal argon laser photocoagulation has proved ineffective in prevention of corneal vascularization (45). Keratoplasty has also limited success, as the limbal blood supply is poor in these patients (45,48).

While it is aimed at reducing ocular irritation and improving vision, Ocular lubricants, therapeutic contact lenses, and lamellar or penetrating keratoplasty have been used (50). Also Transplantation of amnion membrane or limbal stem cells may prove to be a successful concept for the treatment of this disorder (51).

CONCLUSION

The widespread use of SM as an incapacitating warfare agent in the past century has proved its highly long-lasting toxic effects. This experience may ensure further use of the agent in future military conflicts and terrorist attacks. In conclusion, it is shown that there are some severely complicated victims who suffer from eyes, lungs, and skin lesions. Pruritus is the most common complain and a malignant change is the most important lesion, which has to be considered. Mustard gas causes chronic and delayed destructive lesions in the ocular surface and cornea, leading to progressive visual deterioration and ocular irritation. The pathophysiologic features of these changes are not clearly identified. Excised conjunctival and corneal specimens revealed a mixed inflammatory response without any specific features. Based on the clinical appearance of the lesions and the histopathologic findings, an immune-mediated component seems possible.

There is also little information regarding the medical management of acute and delayed toxic effects of SM poisoning, a subject which greatly challenges health care specialists.

As progress continues in strategizing how to counter biologic and chemical threats, it is worthwhile to ponder the words of the economist E. F. Schumacher, who observed ''It is of little use trying to suppress terrorism if the production of deadly devices continues to be deemed a legitimate employment of man's creative powers.''

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