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A Rare Case of Accidental Dimethyl Sulphate Poisoning

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Abstract:
Accidental poisoning of workers occurs occasionally in chemical and pharmaceutical industries. Some of the chemicals involved in industrial poisoning are unique. One such case of poisoning due to dimethyl sulphate is described. Dimethyl sulphate is preferred by the industry because of its low cost and high reactivity. The respiratory and gastrointestinal systems are its primary targets. The diagnosis and early intervention of this poisoning is of paramount importance.
We report two cases of varying degrees of exposure to Dimethyl sulphate accidentally, occurring as a result of a single chemical spillage incident in the Bangalore, India.

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I. Introduction:

Dimethyl sulphate $(CH_3)_2SO_4$ (DMS) is a methylating agent which is a slightly odourous, oily liquid used in the industries(1) and laboratories,(2) in the manufacture of antipyretics,(3) and anticholinergics as pharmaceuticals,(4) perfumes, and pesticides. It is also used medically for chemical cleavage of DNA sequences.(5) DMS is highly toxic, corrosive, and has carcinogenic, mutagenic, and teratogenic potential.(6,7) It's vapour density is 4.35 (air=1). The melting point is −32°C; the boiling point is 188°C. At 20°C the vapour pressure is 0.5 mmHg.(8)

Toxicity is manifested initially by mucosal inflammation of eyes, nose, oropharynx, and airways. This can progress to severe airway oedema and necrosis, and non-cardiogenic pulmonary oedema. Other systemic effects include convulsions, delirium, coma, and renal, hepatic, and cardiac failure. All these features make DMS a potential chemical weapon.

When Dimethyl sulphate comes in contact with a moist mucosal surface, it is hydrolyzed into sulphuric acid, methanol, and methyl hydrogen sulphate. Neurotoxicity occurs due to systemic absorption of methanol. The other two substances formed cause severe irritation and corrosion of mucosa.(9) Absorption occurs readily through the skin, mucous membranes, and gastrointestinal tract. After acute exposure, symptoms are usually delayed for several hours, allowing potentially fatal exposures to occur before the patient becomes aware of their plight.(6,7,10,11)

II. Case Report:

The exposure occurred in a perfume manufacture industry in November 2017. Two male workers aged 30-40 years were in a workshop wearing overalls, boots, and gloves, but no other protective gear.

One to two hours after exposure both workers noted eye irritation with discomfort, lacrimation, and erythema. Twelve hours after the exposure, both the patients had rhinorrhoea, and a burning ocular foreign body sensation and burning sensation in chest.

The patients were admitted in the hospital for observation for possible development of delayed non cardiogenic pulmonary edema.

The patient eyes were irrigated with the normal saline and slit lamp examination showed Conjuctival congestion with Chemosis and corneal abrasions in one patient and Conjuctival congestion and chemosis with dilated pupil in other patient. On throat examination it was congested and respiratory examination showed presence of diffuse wheezes for both the patients. Under local anaesthesia, fibreoptic laryngoscopy was performed on both the patients. It revealed normal vocal cords and arytenoids without any laryngeal oedema. Hence both of them were not intubated.

After 1 day of hospitalisation one of the patient desaturated and the patient was then commenced on oxygen inhalation. The patient saturation was monitored throughout the day and it was normal after inhalation.

A mild transient increased leucocyte count with neutrophilia was seen only in one of the patient with other blood tests, chest radiograph, and an electrocardiogram was all normal.

PFT showed mixed ventilator defect with predominant obstructive defect for one patient and normal spirometry in other patient.

The patient was then commenced on IV antipseudomanal pencillins, nebulisation with budesonide and salbutamol, IV proton pump inhibitors, Steroid eye ointment, cycloplegics and other supportive measures.

Both of them were monitored closely with resolution of wheezes and burning sensation of eyes by day 4 of hospitalisation. As the progress in the hospital was uneventful, both of them were discharged in stable condition.

After discharge both of them were followed up on Outpatient basis every week and both of them returned to their normal work with routine activities after 4 weeks.

III. Discussion:

As the Dimethly sulphate was a fatal chemical, all the physicians should be aware of the active line of management. The patients who were most exposed to Dimethyl sulphate developed symptoms of toxicity more quickly, and their symptoms were worse and longer lasting. The mucous membrane injury initially developing in the eyes and then progressing to nasal and upper airway symptoms is characteristic. The raised white cell count is also characteristic. Fortunately none of our cases developed the severe laryngeal oedema, non-cardiogenic pulmonary oedema, toxic shock, encephalopathy, myocardial damage, or methanol toxicity that can occur with greater exposures.

General treatment of DMS toxicity involves minimising exposure of patients and decontamination, together with close observation and supportive management. Early initiation of antibiotics and steroid nebulisation decreases the incidence of secondary infections and non cardiogenic pulmonary oedema. Ethanol administration or haemodialysis may be undertaken if there is evidence of methanol toxicity.(9)

All the Workers employed in the industries utilizing this hazardous chemical should use protective clothing, rubber gloves, and eye protective wear to prevent any accidental exposure. Exposed workers must be immediately subjected to first aid like washing before transferring to a nearby hospital.

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