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EVALUATION OF ACUTE CASES OF SO_2 GAS POISONING DUE TO REACTION OF SODIUM HYDROSULFITE WITH WATER

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Abstract
Solid sodium hydrosulfite is a very reactive chemical, which can decompose when exposed to air and moisture or in contact with small amount of water. Decomposition will result in spontaneous ignition and liberation of toxic gas- Sulphur dioxide (SO2).

In present article, it is aimed to discuss the effects of SO2 released through air because of the unconsciousness mixture of a sodium hydrosulphite contained compound with water to the guidance of the current literature. In a textile factory of Diyarbakir province, adding water to the plastic can of sodium hydrosulphide used for removing the points of the clothes revealed SO2 gases to the environment and 12 personnel were effected by the way of inhalation.

The effects of SO2 gases on humanbeings are depend on the intensity and the time of duration. The personnel worked in industrial and textile factories have to be informed

about these kinds of chemical reactions. The prevention and first aid and the technical hardware have to be provided.

Key words: Sodium hydrosulphite, poisoning, sulphur dioxide, inhalation, environment

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Introduction

Sodium dithionite (Molecular formula: Na2O4S2) synonyms: hydrolin, sodium sulfoxylate, sodium hydrosulphite, CAS No: 7775-14-6. Sodium hydrosulfite in solid white crystalline powder form is a very effective reducing agent. It has wide applications in various industries, including textile, pulp and paper, kaolin clay, and water treatment. Solid sodium hydrosulfite is highly stable when stored in dry closed containers. However, solid sodium hydrosulfite is a very reactive chemical, which can decompose when exposed to air and moisture or in contact with small amount of water. Decomposition will result in spontaneous ignition and liberation of toxic gas- Sulphur dioxide (SO2) (1,3).

As it is highly reactive, SO2 has a highly non-uniform dose distribution along the conductive airways of the respiratory tract. For low to moderate tidal volumes and nasal breathing, the penetration into the lungs is negligible. For larger tidal volumes and oral inhalation, doses of interest may extend into segmental bronchi. SO2 can only reach the gas-exchange region of the lungs after sorption onto fine particles; and the available particle surface is limited except when very large mass concentrations of fine particles are present (WHO 1987; WHO 1994a). Another special consideration for SO2 is that there is a great variation in susceptibility to a bronchoconstrictive response (2-4). In present article, we aimed to discuss the effects of SO2 released through air because of the unconsciousness mixture of a sodium hydrosulfite contained compound with water to the guidance of the literature.

Materials and methods

In a textile factory of Diyarbakir province, adding water to the plastic can of sodium hydrosulfite used for removing the points of the clothes revealed SO2 gases to the environment and 12 personnel were effected by the way of inhalation.

Physical examinations, arterial blood gases (ABG), Posterior-anterior (PA) lung graphies, electrocardiography (EKG) and biochemical examinations were performed our patients in the emergency department and they were treated and took under observation if necessary.

Results

The whole patients applied to the emergency department were man and their average ages were 28.18±4.05 (range from 23-37). Breathlessness was the common symptom in our patients. Physical examinations revealed conjunctival hyperemia and

bronchoconstriction in two of the patients. The ABG parameters of the patients were between the normal ranges.

The ECG changes were atrial fibrillation, tachicardia and premature beat in one patient whereas T- changes in another. 100 % oxygen and salbutamol 200 mg were administered to all patients applied to the emergency department.

Patients observed in emergency room for 24 hours. During the observation, physical examinations, PA lung graphies, ECG's and ABG's of the patients were controlled and any pathologic event didn't develop. In the control ECG of the patient who had atrial fibrillation, tachicardia and premature beats, it was seen that the heart rate become normal and the premature beats were slow down, but it was notice that the patient had had atrial fibrillation previously. The control ECG of the patient who had T- changes was normal (Table I).

Discussion

SO2 is a colorless gas with a pungent irritating odor, which is noticeable at 3-5 ppm. It is employed in the manufacture of sulfuric acid and is a potential occupational hazard in paper mills, steel works and oil refineries. The combustion of fossil fuels for heating and power generation is the major source of SO2 in the environment. SO2 is highly water-soluble and so is efficiently absorbed in the upper respiratory tract. The irritant effects of SO2 are thought to be caused by the rapidity with which it forms sulphurous acid on contact with moist mucous membranes. Ammonia present in the oral and nasal passages has an immediate neutralizing effect on low concentrations of inhaled SO2 with the formation of sulfite or bisulfate. The metabolic fate of these species is not fully understood though absorbed SO2 is eventually excreted as sulfate (4-9).

Table 1: The demographic values of patients.

		Patients
Gender	Male	12
Complaint in Admission	Breathlessness **Coughing** Nausea-Vomiting **Chest pain** Sore throat	12 **1** 1 **1** 1
Physical examination	Conjunctival hyperemia Pulmoner examination **- Normal** - Low respiration sound	2 **10** 2
X-ray	PA Pulmoner radiography **- Normal** - Abnormal	 **11** 1
EKG*	**Normal** Tachicardia, dysrrhythmia **ST-T changes**	**10** 1 **1**
ABG**	Normal **Abnormal**	12 **-**
Treatment	Oxygen+Salbutamol	12

Following exposure to 5 ppm SO2, dryness of the nose and throat and a measurable increase in the resistance to bronchial airflow is observed; a decrease in tidal volume occurs at 6-8 ppm. Lacrimation, rhinorrhoea, cough, increased bronchial secretions and bronchoconstriction occur at concentrations exceeding 10-50 ppm. In more severe cases, non-cardiac pulmonary edema and respiratory arrest may supervene. 400-500 ppm is considered to be an immediate danger to life and 1000 ppm can lead to death within 10 min. Corneal and dermal burns can follow exposure to liquefied SO2. Survivors of massive SO2 exposure have shown a chronic obstructive defect in serial pulmonary studies along with bronchial hyper reactivity (10-11).

In present study the symptoms of the upper respiratory tract such as breathlessness, coughing, sore throat and nausea vomiting were seen. Only two patients had conjunctival hyperemia and bronchoconstriction. Non-cardiac pulmonary edema didn't develop in our patients. We could not measure the amount of SO2 in the environment but it was estimated that patients exposed to approximately 20-ppm SO2. Two of the patients exposed to the gases for 30 minutes while the others exposed for nearly one hour but they

were working in farther place. The patients applied to the emergency department 30 minutes after the event.

After removal from exposure, symptomatic patients should be assessed at a hospital. Admission for observation and treatment is mandatory in those with persisting symptoms following substantial SO2 exposure. The eyes and skin should be irrigated with water, if irritation is present. Corneal and dermal burns from liquefied SO2 should be treated conventionally and with the involvement of ophthalmic and plastic surgeons respectively. Nebulized salbutamol or other B2 agonist can relieve bronchoconstriction; inhaled corticosteroids may be of value. The role of corticosteroids (inhaled or systemic) in preventing the onset of non-cardiogenic pulmonary edema is uncertain. Mechanical ventilation with positive end-expiratory pressure (PEEP) will be required if non-cardiogenic pulmonary edema ensues (6,10). Oxygen and 200 mg salbutamol mediflexes were administered to the patients applied to the emergency department. In our cases, SO2 gases released from adding water unconsciously to sodium hydrosulfite caused toxic effects by the way of inhalation. It was a fault that the patients hadn't been educated for the reactions. The personnel worked in industrial and textile factories have to be informed about these kinds of chemical reactions. The prevention and first aid and the technical hardware have to be provided.

Conclusion

One of the sources of SO2 gases that is toxic to human beings and causes air pollution is sodium hydrosulphite. The effects of SO2 gases on human beings are depend on the intensity and the time of duration. The personnel worked in industrial and textile factories have to be informed about these kinds of chemical reactions. The prevention and first aid and the technical hardware have to be provided.

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