SL 036586

CHEST PAIN AND HYPOXEMIA FROM INHALATION OF A TRICHLOROETHANE AEROSOL PRODUCT

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ABSTRACT

A 25-year-old man developed severe shortness of breath, constricting chest pressure, chest pain, cough and myalgia following acute exposure to a waterproofing aerosol that contained trichloroethane. He became febrile and developed a small area of atetectasis with significant hypoxemia. Recovery was complete within 36 hours. This experience suggests that casual use of a trichloroethane aerosol with a surface active agent can cause acute pulmonary toxicity. The mechanism of this injury is unknown.

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 0731-3810/84/2004-0333$3.50/0

TRICHLOROETHANE AEROSOL

The San Francisco Bay Area Regional Poison Control Center (SFBAPCC) has received several inquiries regarding the toxic effects of waterproofing aerosol products. These aerosol products contain a common solvent, 1-1-1-trichloroethane (methylchloroform, alpha-trichloroethane). Fatal cases (1,2,3,4,5,6,7) of depression of the central nervous system from inhalation of trichloroethane vapors have been reported, but direct respiratory toxicity is not a well-recognized feature of trichloroethane intoxication. A patient in whom chest pain and hypoxemia developed after use of an aerosol trichloroethane product is described and the experience of the SFBAPCC with eight other patients in whom respiratory symptoms developed after inhalation of the same product is summarized.

CASE REPORT

On January 4, 1982, a 24-year-old white man treated a pair of pants with a waterproofing aerosol in his bathroom. Three hours later, he was admitted to a local emergency room because of severe shortness of breath, constricting chest pressure, substernal chest pain on deep breathing, cough, and myalgias. Arterial blood measurements obtained while the patient breathed room air were as follows: pH 7.44, PO_2 58 mmHg, PCO_2 34 mmHg, and bicarbonate concentration 23 mEq/L. A chest roentgenogram showed no abnormalities. His temperature was 98°F, blood pressure was 110/60 mmHg, pulse rate was 104 beats/minute, and respiratory rate was 24 breaths/minute.

Physical examination revealed a healthy man in respiratory distress. Conjunctiva and oropharynx showed no abnormalities. Occasional rales were heard over both lungs. Heart sounds were regular and of good quality. No murmurs were heard. Electrocardiogram was not performed. The patient had an unsteady gait but no other neurologic or other abnormalities. White blood cell count was 14,800/cu mm with a normal differential count, hemoglobin was 15.3 gm/dl, and hematocrit was 44.5%. Results of urinalysis were within normal limits. Repeat arterial blood measurements while on oxygen, 6 liters/minute, administered via nasal cannulae were as follows: pH 7.42, PO_2 70 mmHg, PCO_2 44 mmHg, and

bicarbonate concentration 27 mEq/L. The patient was transferred to the respiratory intensive care unit.

His temperature rose to 100°F during the night and a second chest roentgenogram showed a small patch of linear atelectasis in the lingula of the left upper lobe; however, chest pain, cough, and dyspnea had abated. The temperature returned to normal by the next morning. The patient was discharged 36 hours after admission. Follow-up examination and roentgenogram one week later showed no abnormalities.

DISCUSSION

The medical literature describing the experimental use of trichloroethane as an inhaled anesthetic gas (8), as well as reports of accidental deaths due to inhalation of trichloroethane vapor, notably lacks evidence that trichloroethane is a serious irritant of the respiratory tract. Adams et al (9) demonstrated an acute LD^{50} in rats at exposures of 18,000 ppm for 3 hours and 14,000 ppm for 7 hours. The cause of death was progressive respiratory depression but without evidence of direct pulmonary parenchymal damage. Repeated 7-hour exposures of rats, guinea pigs, and rabbits to 5,000 ppm resulted in mild narcosis but no lung, liver, or kidney injuries or deaths. Successful surgical anesthesia has been produced in human subjects with inspired trichloroethane concentrations of 20,000 to 30,000 ppm (10). Recovery from light plane anesthesia is 3-5 minutes. The incidence of post anesthesia nausea, vomiting, and fatigue is low with no reports of pulmonary irritation.

In human studies, transient mild irritation of the eyes and minimal impairment of coordination occurred after exposure to 900-1,000 ppm; loss of equilibrium, headache, and lassitude were experienced at levels of about 1,700 ppm (11). Subjects exposed to an increasing concentration of trichloroethane at a constant rate for 15 minutes, peaking at 2,650 ppm, were unable to stand unaided. The current threshold limit value for industrial exposure to trichloroethane is 350 ppm (13).

Over 30 deaths and accidental intoxications from trichloroethane inhalation have been reported (1,2,3,4,5,6,7,14,15,16,17). The majority of these cases occurred when the liquid solvent was used in a ined space

or poorly ventilated area. Trichloroethane concentrations exceeding several thousand ppm were reached on many occasions. All of the deaths were due to depression of the central nervous system with resultant apnea and peripheral vascular collapse. The earliest symptoms of intoxication were dizziness and lassitude without symptoms of pulmonary irritation (17).

During the winter months of 1981-1982, the SFBAPCC received nine reports of adults (Table I) who developed respiratory symptoms after use of an aerosolized waterproofing product which contained 96.6% trichloroethane, 2.4% fluorocarbon resin, and a liquid cholesterol emulsifier, 1% ritachol (a registered trademark of the R.I.T.A. Corporation). The propellants in the product were isobutane and propane. The persons affected usually had been spraying this agent on an article of clothing or shoes for five to fifteen minutes in a small, poorly ventilated area, such as a bathroom or bedroom. Within a few hours, substernal chest discomfort and pain on deep inspiration developed in all patients; eight also complained of coughing. Six of nine patients noted significant shortness of breath. Several noted drowsiness initially and myalgias or lethargy developed in some subsequently. Symptoms resolved spontaneously within the ensuing 24 hours with the exception of the patient reported here and Case #7.

Hall and Hine (3) reported two provocative cases of fatal trichloroethane intoxication. One was a 29-year-old man who was found dead with a trichloroethane-laden cloth over his nose and mouth. An autopsy revealed acute pneumonitis that the investigators suggested may have been caused by aspiration of the trichloroethane liquid. The second was a 19-year-old woman who had been reported sniffing a cleaning fluid for several days before she was found dead. Microscopic examination showed the bronchi contained denuded epithelium.

Trichloroethane is a chlorinated aliphatic hydrocarbon and can be irritating to mucous membranes. Ocular exposures to trichloroethane cause transient conjunctival discomfort without corneal injury in animals and humans (20,21).

A recent experiment in rats by Dickerson and Biesemeir (18) suggests that trichloroethane liquid instilled directly into the airways is capable of causing lung injury. We have observed that an aerosol preparation of

TABLE I. Trichloroethane Adult Case Summary

Case No.	Inhalation Time (mins)	PCC Contact Post-Exposure (hrs)	Symptoms	Comments
1	15	5	Cough, shortness of breath, inability to take a deep breath.	Patient had underlying asthma, used bronchodilater inhaler more frequently than usual. Symptoms resolved in 12 hours.
2	5	3	Nausea, dizziness, cough productive of white sputum.	Patient saw physician 2 days later. Examination, chest X-ray, white blood cell count were normal. Symptoms much resolved.
3	10-15	3	Nausea, headache, chills, shortness of breath, *continuous* cough, painful deep breathing.	Patient went to local emergency room. Chest X-ray and arterial blood gases were normal, white blood cell count was 14,400/cumm.
4	5-10	2	Faintness, dizzy, weak, cough, pain on deep inspiration.	No follow-up.
5	10-15	18	Severe shortness of breath.	Patient went to local emergency room.
6	10-15	12	Woke in a.m. with cough and chest discomfort.	Patient went to local emergency room.
7*	60	16-18	Headache, upset stomach, difficulty breathing, hemoptysis.	Patient went to local emergency room. Chest X-ray was abnormal. Given diagnosis of allergic alveolitis and treated with prednisone.
8**	15	3	Severe shortness of breath, cough, chest tightness, pain on inspiration.	Patient went to local emergency room. Chest X-ray showed left upper lobe atelectasis. Arterial blood gases with PO_2 58 mmHg.
9	Sprayed article left in bedroom all night	14-18	Woke in a.m. with cough and shortness of breath.	Patient contacted local emergency room.

*Medical record could not be obtained. **Case reported here.

trichloroethane can cause symptoms of pulmonary irritation, whereas trichloroethane vapor apparently does not cause significant injury to the respiratory tract (19). A possible explanation is that inhalation of the trichloroethane aerosol preparation leads to higher local concentrations of trichloroethane at the respiratory mucosa than does inhalation of the vapor. Trichloroethane is highly soluble in organic solvents, but it is only slightly soluble in water (11). After inhalation of trichloroethane vapor, little would deposit anywhere in the airways, but systemic absorpiton could occur in the alveoli because of the large alveolar surface area and the lipid layer that lines the alveoli. On the other hand, after inhalation of trichloroethane aerosol, airway deposition of aerosol droplets might result in high local concentrations of trichloroethane in the upper airway.

All of our cases in this report were associated with one particular waterproofing product that contained a surface active agent, a blend of mineral oil and lanolin alcohol. Lanolin alcohol is principally composed of cholesterol, dihydrocholesterol, and lanosterol. Such a formulation would permit trichloroethane to be more water-soluble and might thus result in increased contact between inhaled trichloroethane and airway epithelial cells. The surface active agent, ritachol, has yet to be implicated as a respiratory tract irritant. It is nontoxic when ingested and direct application to the eye or skin have not produced irritation or injury. The concentration of the surface active agent is very low and the amounts inhaled would unlikely be sufficient to cause direct lung injury. However, an allergic sensitivity to any of the individual agents cannot be excluded in our patients. Fluorocarbon resins have not been shown to be irritants to the skin and eyes; inhalation of these compounds by laboratory animals have not produced respiratory complications. Moreover, other coincident conditions could explain the symptoms reported in some of the patients. Acute pulmonary embolus was not excluded in the evaluation of our index case. The patient's age, rapid resolution of symptoms, normal findings on heart examination, and the absence of cardiac murmurs make the possibility of pulmonary embolus unlikely in the case. The similarity of sympatoms experienced in the nine patients and temporal relationship of exposure to a single waterproofing aerosol at high concentrations suggest that the pro[illegible]was responsible for the symptoms.

SUMMARY

We report a patient who experienced tachypnea, pleuritic chest pain, hypoxia, and tachycardia following the use of an aerosolized trichloroethane product. The patient had significant hypoxemia which necessitated hospitalization. The hypoxemia may hav been due to atelectasis. A small area of linear atelectasis was seen on a second chest X-ray examination approximately 12 hours after admission. A follow-up X-ray examination one week after the patient's discharge was normal. The patient had used a waterproofing product in a poorly ventilated and enclosed space for approximately fifteen minutes. The unsteady gait se n on the patient's arrival at the emergency room is also suggestive of a significant inhalation exposure to high concentrations of trichloroethane.

This experience suggests that casual use of aerosolized trichloroethane combined with a surface active agent may produce considerable acute respiratory distress. This formulation increases the water solubility of trichloroethane and enhances its deposition in the upper airway to produce symptoms reported here. It is unlikely that the pulmonary irritation can be attributed to either the trichloroethane, the surface active agent, or propellant alone.

ACKNOWLEDGEMENT

We are grateful to Drs. Edward K. Blasdel and Winston Lee for permission to review the patient's medical records, the R.I.T.A. Chemical Corporation, and the staff of the San Francsico Bay Area Regional Poison Control Center for their cooperation in collecting the information.

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