Methylene Chloride:
Report of Five Exposures and Two Deaths

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Five patients presented to the emergency department (ED) following exposure in an enclosed space to methylene chloride (dichloromethane), used for removing paint. Two workers and three rescuers were involved. Two rescuers complained only of dizziness and mild nausea, and were subsequently discharged from the ED. One rescuer was asymptomatic. Worker no. 1 arrived in cardiac arrest and eventually died in the ED despite resuscitation efforts. Worker no. 2 also presented to the ED in cardiac arrest, and was successfully resuscitated to pulse and blood pressure. However, he never regained consciousness or spontaneous respirations, and died on the fourth day. Of interest is that worker no. 2's carboxyhemoglobin level increased from 2% to 8% over the 9 hours following admission, despite administration of 40% to 50% oxygen by endotracheal tube. Among the conclusions that can be drawn are (1) the cause of death in these patients was not carbon monoxide poisoning, but solvent-induced narcosis; (2) carboxyhemoglobin levels may continue to rise following cessation of exposure, despite administration of high flow oxygen; (3) rescuers can easily become victims if proper protective clothing and respirators are not worn. (Am J Emerg Med 1990;8:534-537. © 1990 by W.B. Saunders Company.)

Methylene chloride (dischloromethane; CH₂Cl₂) is one of the most widely used solvents in home and industry. It is utilized in aerosols, fire extinguishers, fumigants, and paint removers. Of interest is that about 635 million pounds of methylene chloride were produced in 1980, and that about 30% of this total was used in the production of paint stripping compounds. While it has been considered to be one of the safest of the chlorinated hydrocarbon solvents, several fatalities due to acute exposures to high concentrations have been documented in the literature.

The following report details an episode of acute exposure involving five victims, including two fatalities. These cases demonstrate some of the unique properties, clinical effects, and hazards of this chlorinated hydrocarbon compound.

CASE REPORT

Five patients presented to the Northwestern Memorial Hospital (Chicago, IL) emergency department (ED) after being exposed in an enclosed space to a methylene chloride-based compound used for removing paint. Paramedics had been called to the scene after building security personnel pulled two unconscious men from a small washroom, where there was a strong chemical odor and an open container of paint stripper labeled “Seal-Off” (Sanitek Products, Inc; Los Angeles, CA). This product contains 91.2% methylene chloride and 6.0% methanol. Both men were last seen alive 20 to 30 minutes prior to being discovered unconscious. Patient no. 1, one of the two initial victims, was wearing a half mask with organic vapor cartridges. Intertwined limbs suggest that one of the two initial victims was trying to remove the other from the room before both succumbed. This made it difficult for the security personnel to separate the victims and two of the three rescuers also became symptomatic. Both initial victims (patient no. 1 and no. 2), were in cardiac arrest; one of the rescuers (patient no. 3) who had performed mouth-to-mouth respiration on the two victims was complaining of nausea and vomiting.

Patients

Patient no. 1, a 29-year-old male smoker, was in asystole with no evidence of obvious trauma. Areas of purple discoloration or lividity were noted on the forehead and left hand without blistering or skin breakdown. Following verification of endotracheal tube placement, advanced cardiac life support (ACLS) procedures were performed without response. Arterial blood gas (ABG) values on ED arrival are shown in Table 1. Patient no. 1 was pronounced dead 15 minutes after arriving at the ED.

At autopsy, mild pulmonary edema with hematomas was noted. Skin burns were also evident. Urine drug screen was negative for cocaine and alcohol and the postmortem carboxyhemoglobin level was 6%. The serum methylene chloride level was 15.5 mg/dL. Methylene chloride was also present at 22 µg/mL in the urine, 35 µg/g in the liver, 109 µg/g in the brain, 93 µg/g in the spleen, 58 µg/g in the kidneys and 40 µg/g in the lungs.

Patient no. 2, a 32-year-old male worker, was found unconscious in the room with patient no. 1. At the scene this patient was initially reported by paramedics to be in pulseless idioventricular rhythm (rate 40/minute). There was no response to prehospital cardiopulmonary resuscitation (CPR) or ACLS procedures. The initial ED examination showed a pulseless, apneic patient with fine ventricular fibrillation on cardiac monitoring. The endotracheal tube was improperly placed in the esophagus and was repositioned. The patient was noted to have purple discoloration “ecchymosis or lividity” on the forehead and right arm, with no other evidence of injury. ED treatment included cardiac defibrillation and routine ACLS recommended drugs with the exception of more frequent epinephrine doses. The cardiac rhythm changed from fine ventricular fibrillation, to asystole, to electrical-mechanical dissociation, to idioventricular rhythm with a rate of 80/minute with palpable pulses and a corresponding blood pressure of 90 mm Hg (palpable) systolic after 19 minutes in the ED. A total of 6 mg of epinephrine, 2 mg of atropine along with dopamine were administered during the resuscitation. Vital signs quickly stabilized prior to transfer to the intensive care...
unit, but the patient at no time demonstrated signs of awakening or spontaneous respirations.

In the intensive care unit, the patient remained unresponsive and required mechanical ventilation. Arterial blood gases (Table 1) showed gradual improvement of respiratory and metabolic acidosis with controlled respirations ventilator and administration of sodium bicarbonate. Carboxyhemoglobin levels were low initially (0% to 2%), and rose to a maximum of 8% at 8 hours after admission (Figure 1). Liver enzyme elevations were also noted: serum lactate dehydrogenase, 4,300 U/L; aspartate aminotransferase, 4,350 U/L; alkaline phosphatase, 328 U/L. Urine drug screen was positive for cocaine and ethanol upon admission into the ED. Electroencephalogram (EEG) showed no brainstem activity at 18 hours after admission (Figure 1).

Autopsy was essentially unremarkable except for a right lower lobe pneumonia with consolidation, and cerebral edema with tonsillar herniation. Methylene chloride levels were negative in the brain tissue and urine at autopsy.

**Rescuers**

Patient no. 3 was a 38-year-old male security guard with a history of smoking, who helped remove the two victims from the bathroom and subsequently performed mouth-to-mouth resuscitation on patient no. 1. He was administered 100% oxygen via nonrebreathing facemask for 3 hours and was then discharged to home. At 2-week follow-up, she complained of intermittent headaches, nausea, and abdominal pain.

Patient no. 5 was the person who discovered the two initial victims and assisted in removing them from the room. He had no symptoms other than extreme emotional upset, consented to a carboxyhemoglobin determination, but refused to sign into the ED as a patient.

**DISCUSSION**

Because of its almost ubiquitous use in home and industry, it is not surprising that there are several reports of morbidity and mortality involving methylene chloride. One of the first reports (1952) involved four exposures with one fatality at a plant involved in the manufacture of oleoresin. While the authors noted the acute narcotic and irritant actions of this solvent, they had hypothesized an effect on the hemopoietic system as an acute cause of injury.

Over the succeeding 37 years, however, it became increasingly apparent that methylene chloride was substantially different than other chemically similar solvents. In the early 1970s, endogenous generation of carboxyhemoglobin following inhalation of methylene chloride was described, since which time contribution of carbon monoxide to the toxic effects noted in acute and chronic methylene chloride poisoning has been under debate.

This tragic episode demonstrates several points regarding methylene chloride toxicity that has not been previously well-documented in the literature. While the use of respirators with organic vapor cartridges has been previously advocated to minimize toxic exposure, it appears that appropriate ventilation in confined spaces is the most important determinant. It has been estimated that at a concentration of 1,000 ppm methylene chloride, the service life of a standard organic vapor cartridge is only 15.8 minutes before a breakthrough of 100 ppm methylene chloride occurs. Chin-type canisters and chemical cartridge respirators with small volumes of sorbent are therefore not recommended for prolonged use in a methylene chloride-contaminated atmosphere. In a review of 118 industrial solvent exposures in which resolved over 20 minutes. Physical examination was unremarkable; ABGs and a carboxyhemoglobin level were obtained (Table 1). The patient was administered 100% oxygen via nonrebreathing facemask for 3 hours and was then discharged to home. At 2-week follow-up, she complained of intermittent headaches, nausea, and abdominal pain.

**TABLE 1. ABG of the Patients**

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**ABBREVIATIONS:** RA, room air; HbCO, Carboxyhemoglobin level.
Great Britain, of 53 people rendered unconscious, 40 were working in confined spaces, while only 30 of 76 less affected patients were working in confined spaces. Furthermore, of the 33 workers exposed to methylene chloride in this series, 8 of the 13 (62%) unconscious workers were working in confined spaces while only 6 of 20 (30%) conscious workers were working in confined spaces.2

A steady increase in carboxyhemoglobin levels can occur following cessation of exposure despite administration of supplemental oxygen even though the physiological consequences may be minimal. It has been estimated that an exposure to about 100 ppm methylene chloride is equivalent in a 50 ppm exposure to carbon monoxide.1 In patient no. 2, carboxyhemoglobin levels rose from 2% on admission to 8% about 9 hours later. Stewart noted a rise in carboxyhemoglobin from an average of 4.3% to a peak average of 7.0% in four subjects following exposure of methylene chloride in the setting of home ventilation.3 Additionally, carboxyhemoglobin levels as high as 50% following methylene chloride exposure have been rarely documented.10 A recent review of 24 acute exposures (13 domestic and 11 industrial) over a 50-year period noted that only 4 of the 21 acutely symptomatic patients were treated with oxygen.11 Modest elevations of carboxyhemoglobin levels have been noted in patients exposed to methylene chloride; in patient no. 2 this increase occurred despite administration of high flow normobaric oxygen.3,10-12 Additionally, while hyperbaric oxygen has recently been reported to be of some potential benefit in methylene chloride exposure, its administration did not prevent elevations of carboxyhemoglobin in the two patients described by Rioux and Meyers.13

A delayed rise in carboxyhemoglobin levels following the cessation of methylene chloride exposure can be explained by endogenous conversion to carbon monoxide by the liver microsomal system.14 This endogenous production of carbon monoxide is thought to be so significant that a 60-year-old worker is believed to have developed dementia secondary to the carbon monoxide produced by his exposure to methylene chloride over a 20-year period.13 The rising carboxyhemoglobin levels in patient no. 2 over an 8-hour period the slow decomposition of carboxyhemoglobin is consistent with previous case reports of carboxyhemoglobin.16-18 Additionally, the fact that patient no. 2 did not have skin decontamination may also have contributed to this phenomenon.17

Solvent induced narcosis predominates over the generation of carbon monoxide as the acute cause of death. The relatively rapid onset of central nervous system effects in patients presented here, together with similar effects described in other case reports of fatalities, is consistent with the solvent itself being the cause of death rather than the relatively low levels of carbon monoxide generated.3,5,19

Chemical burns caused by methylene chloride may appear as livor mortis. Post mortem lividity usually becomes apparent on the dependent portion of the body 1 to 2 hours after death.20 In patient no. 1, it appeared to be present within 1 hour after he was last seen alive. Additionally, the purplish suggillations are consistent with asphyxiation being the mode of death rather than carbon monoxide poisoning (which gives cherry-red suggillations).20

Caution should be exercised, however, in the identification of these postmortem changes, as erythema induced by heat injuries can cause findings similar to that of livor mortis.21 A false impression that victims may have been in arrest for hours rather than minutes may lead to less vigorous or lack of initiation of resuscitation. Methylene chloride has been known to cause second and third degree burns upon direct contact in similar situations.22 The methylene chloride levels obtained post mortem in patient no. 1 and during hospitalization in patient no. 2 are somewhat lower than in previously described fatal cases. Stevenson et al analyzed blood concentrations of methylene chloride in 18 subjects exposed to an atmosphere of 450 ppm of methylene chloride—approximately the exposure of spray painters are in domestic settings.23 The blood concentration appeared to peak at the first hour postexposure at an average methylene chloride level of 370 ppb (0.0370 mg/dL) for exposed nonsmokers and 414 ppb (0.0414 mg/dL) for exposed smokers. These blood levels were estimated to be approximately 1/1000 of the exposure level in ambient air. The Occupational Safety and Health Administration (OSHA) has estimated that the atmospheric concentration of methylene chloride in this case was 4.5% (45,000 ppm) to 14% (140,000 ppm). According to Stevenson's calculation, this would translate to an estimated serum methylene chloride level of 4.5 mg/dL to 14.0 mg/dL, which was in the range found in patients no. 1 and no. 2 reported here. In the only other level obtained post mortem, Winek reported a level of 29.8 mg/dL in a worker who died while degreasing a tank.19 Of particular interest is that in all three instances (patient no. 1, patient no. 2, and the description by Winek), the workers' foreheads were found immersed in the solvent, thus possibly increasing the concentration of the solvent inhaled.

Rescuers can easily become victims if proper precautionary procedures are not followed. Proper evacuation procedures such as, air supplied or a self-contained positive-pressure breathing apparatus, gloves, gowns, goggles, safety line, and ventilation will prevent mass exposure of victims and rescuers alike.24,25 NIOSH (1985)26 recommends either: (1) any self-contained breathing apparatus with full face piece and operated in a pressure-demand or other positive-pressure mode, or (2) any supplied-air respirator with a full face piece and operated in pressure-demand or other positive-pressure mode, in combination with an auxiliary self-contained breathing apparatus operated in pressure demand or other positive-pressure mode. Although very brief exposure (1 to 2 minutes) as in this case seems to carry minimal risk of serious toxicity, it may be reasonable to monitor the clinical status of rescuers especially following domestic exposures. Industrial exposures might present a greater risk to improperly equipped rescuers, because of the potential for use of larger amounts of solvent.

REFERENCES

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