CASE REPORT

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Two Fatal Cases of Dichloromethane or Chloroform Poisoning

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ABSTRACT: The two cases presented here involve deaths that were due to dichloromethane or chloroform poisoning. The dichloromethane and chloroform were determined in biological specimens by headspace gas chromatography with mass spectrometric detector. In one case, dichloromethane concentrations found were 252 mg/L (blood), 75 mg/kg (brain) and 30 mg/kg (heart). From the case investigation, it was determined that the death was accidental but related to the dichloromethane poisoning. In the other case, chloroform concentrations were 60 mg/kg (blood) and 14 mg/kg (lungs), respectively. This case, the cause of death was chloroform poisoning by forced inhalation in addition to oronasal obstruction.

KEYWORDS: forensic science, dichloromethane, chloroform, poisoning, headspace GC, mass spectrometic detector

Dichloromethane (methylene chloride) is commonly employed commercially and industrially as an aerosol propellant, paint stripper, degreaser, fat extractant and solvent. Toxic reactions to dichloromethane have been limited for the most part to acute exposures, and have resulted from its direct central nervous system depressant effects, its in vivo conversion to carbon monoxide, or its oxidation to phosgene in an open flame (1). About 40% of an absorbed dose of dichloromethane is not eliminated in the exhaled air. A portion of this retained amount is known to be metabolized to carbon monoxide: the half life of excretion of the carbon monoxide produced is approximately 13 hours or 2.5 times of the time need for inhaled carbon monoxide (1,2). This last property is shared with many other chlorinated hydrocarbons. Chloroform has contemporary application in industry and the laboratory as a solvent and chemical intermediate but is historically well known as inhalation anesthetic. Acute chloroform exposure may result in death by respiration failure. The primary effect of chloroform is central nervous system depression with inebriation, anesthesia and narcosis (1,2). The central nervous system depression effect allowed for the nontherapeutic but intentional use of chloroform for recreation, suicide, assault, and homicide. In this report we describe two fatal cases related either dichloromethane or chloroform poisoning.

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Case History

Case # 1

A 51-year-old man, who was the chief executive in a painting and coating factory, was discovered in the TCE (trichloroethylene) tank located at underground of his factory. An interview with employees disclosed that he was seen alive until approximately 12 hours before he was found dead. The scene examination revealed the decedent was lying prone aslant near the entrance of the TCE tank where dichloromethane was used to remove rust from iron sheets.

Autopsy examination showed marked lividity with intense, light-red colored areas on the face, the neck and the anterior chest. Also, Tardieu's spots were appeared on the anterior chest. Internal examination of the brain, kidneys, liver, and lungs revealed prominent organ congestion.

Case # 2

The decedent, a 30-year-old man, was discovered as an abandoned corpse in a river basin. The police arrested the perpetuator. According to the confession from the suspect, he asphyxiated the decedent with an anesthetic—chloroform—and took him in his car. He robbed the decedent of his money and credit card, and abandoned the corpse in a river basin in a rural area. A search of the crime scene discovered one chloroform bottle and a few white paper tissues that were soaked in chloroform.

Autopsy examination was unremarkable save for prominent organ congestion, marked dilatation of lungs and serious arteriosclerosis of the left cardiac coronary artery.

Material and Methods

Analysis of aliphatic halogenated hydrocarbons in biological specimens is generally performed by headspace gas chromatography with flame ionization detector and/or electron capture detector (3–6). In this report, dichloromethane and chloroform contents in biological distribution were determined by head space gas chromatography with mass spectrometric detector.

Human tissue samples obtained at the autopsy were stored at −20 °C until use. Sample preparation were performed as follows: Tissue samples were weighed and homogenized in ice bath. An 1 mL of biological fluids or 1 g of homogenized tissues was mixed with 2 mL of 0.1 N NaCl solution and 50 μL internal standard (0.5% *sec*-butanol). The sample vials were placed in a water bath and maintained at 55°C for 20 min. A 250 μL volume of the

samples prepared in the headspace was withdrawn with a gas tight syringe and injected to the gas chromatography.

Original identification of dichloromethane and chloroform was with a Varian 3400 gas chromatography with flame ionization detector. The columns used was stainless steel column packed with Carbowax 20 M (6 ft × 1/8″). The injector and detector temperatures were 220 and 240°C, respectively. The oven temperature was set at 80°C and held for 3 min, then increased 10°C/min to a final temperature of 180°C. The final temperature was held for 5 min.

Confirmation and quantization of dichloromethane and chloroform was with same chromatographic system interfaced with a Finnigan ITD mass spectrometric detector. The column used was a DB-5, polydimethyldiphenyl 5% siloxane capillary column cut to 30 meters. Helium (4 psi) was the carrier gas. The injector and detector temperatures were 250 and 270°C, respectively. Initial oven temperature was set at 70°C, then the temperature program was set as mentioned earlier. The retention times for dichloromethane, chloroform and internal standard were 2.59, 3.15 and 4.1 min, respectively. We confirmed dichloromethane from the base peak at m/z 49 of [CH_2Cl^+] and m/z 84 of parent ion, and chloroform from m/z 83 of [$CHCl_2^+$] and m/z 118 of parent ion.

Quantization, using a selective ion monitoring program, was based on the peak area ratios of dichloromethane and chloroform to internal standard in comparison to known standard. Dichloromethane contents were detected in blood, brain and other organs from Case # 1. Chloroform was detected in blood and lungs samples of Case # 2.

Results and Discussion

We obtained chromatograms and mass spectra from biological specimens of the decedent were obtained as Fig. 1 for case # 1 and Fig. 2 for case # 2. The following results (Table 1 and Table 2) were estimated from chromatographic data.

The blood sample from Case # 1 contained 3% of carbon monoxide hemoglobin (COHb).

The biological fluids (blood and gastric content samples) were also tested for the presence of ethanol, cyanide, pesticides, sedative hypnotics, barbiturates and antidepressants. None of these compounds were found in both cases.

Case # 1

The narcotic effects of this chemical have been held accountable for only a few fatalities in U.S., some of them were industrial accidents; postmortem blood dichloromethane concentrations of 252 and 298 mg/L in two cases. Chemical finding for a death by inhalation following home usage of a paint remover were 510 mg/L in blood and 144 mg/kg in liver. The COHb level were also detected as 3% of blood hemoglobin content (2). In this case, the postmortem interval was less than one day. The dichloromethane concentration in a biological specimens in this case is lethal level. As well as COHb level, which could be generated from metabolites of dichloromethane, the lethal level of dichloromethane finding in biological specimen is adequate to confirm that death was due to dichloromethane poisoning. This is the first known case of dichloromethane poisoning in Korea.

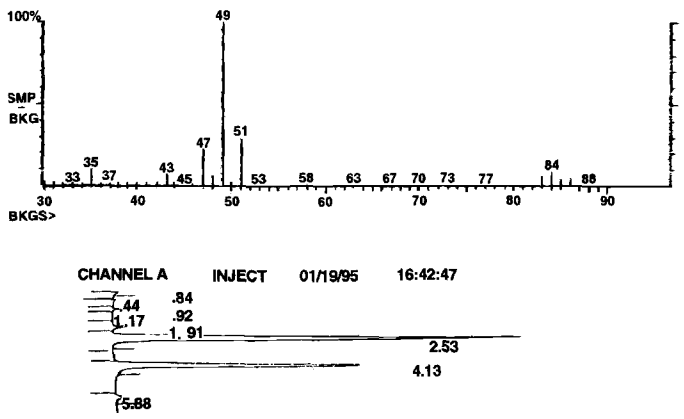

FIG. 1—*Gas chromatogram and mass spectrum of methylene chloride obtained for the blood sample in Case # 1 (methylene chloride: 2.53 min, s-buthanol: 4.13 min).*

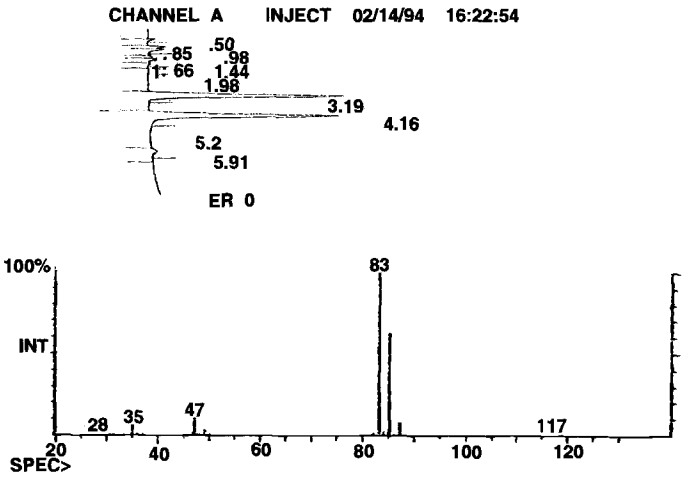

FIG. 2—*Gas chromatogram and mass spectrum of chloroform obtained for the blood sample in Case # 2 (chloroform: 3.19 min s-buthanol: 4.16 min).*

TABLE 1—*Distribution of dichloromethane.*

Specimen	Dichloromethane
Blood	252 mg/L
Gastric Contents	42 mg/L
Kidneys	59 mg/kg
Lungs	26 mg/kg
Heart	30 mg/kg
Liver	56 mg/kg
Brain	75 mg/kg

TABLE 2—*Distribution of chloroform.*

Specimen	Chloroform
Blood	60 mg/L
Lungs	14 mg/kg

Case # 2

Acute ingestion of as little as 10 mL of chloroform may result in death due to central nervous system depression. Exposure to air concentrations of 100–1000 ppm for short periods may cause

discomfort and dizziness, and concentrations of 7000–20,000 ppm will produce rapid loss of consciousness. The many acute fatalities due to the intentional or forced inhalation of chloroform has been reported, the concentrations of chloroform in the blood samples were ranged from 30–180 mg/L (2,7). In this case, only blood and lung sample was available from the autopsy of the decedent. The chloroform levels determined in blood and tissue are known to be lethal. Another considerations other than chloroform poisoning were a suffocation and a lethal arrhythmia secondary to cardiac sensitization to catechol amines during the assault with chloroform. However, we can conclude that the death in this case was due to chloroform poisoning by forced inhalation in addition to physical asphyxia of oronasal obstruction without the contribution of a physical assault, because anatomic evidence of injury was not seen at autopsy and chloroform levels may be high enough to cause death.

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