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Fatality due to inhalation of dimethyl sulfide in a confined space: a case report and animal experiments

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Summary. A man was found dead in a tank where gaseous dimethyl sulfide (DMS) was present. The concentrations of DMS in the blood and tissue samples were measured by gas chromatography. Mice were experimentally exposed to various concentrations (5%–55%) of gaseous DMS in a confined space and the course of death and DMS distribution in the bodies were observed to obtain diagnostic criteria for DMS poisoning. As a result it was considered that the cause of death of the victim was consistent with a combination of DMS poisoning and asphyxia due to a hypoxic atmosphere.

Key words: Gas inhalation – Confined space – Poisoning and asphyxia – Dimethyl sulfide – Gas distribution

Zusammenfassung. Ein Mann wurde tot in einem Tank vorgefunden, welcher Dimethylsulfid (DMS) in gasförmigem Zustand enthielt. Die Konzentrationen von DMS wurden im Blut und in den Geweben mit Hilfe der Gaschromatographie bestimmt. Mäuse wurden experimentell verschiedenen Konzentrationen (5–55%) von gasförmigem DMS in einem geschlossenen Raum exponiert und das Sterbegeschehen und die DMS-Verteilung in den Körpern wurde beobachtet, um diagnostische Kriterien für die DMS-Vergiftung zu erhalten. Für den gegenständlichen Fall wurde angenommen, daß der Tod durch eine Kombination von DMS-Vergiftung und Sauerstoffmangel aufgrund einer hypoxischen Atmosphäre zu erklären war.

Schlüsselwörter: Gasinhalation – umschlossener Raum – Vergiftung und Erstickung – Dimethylsulfid – Gasverteilung

Introduction

When a person dies in a confined space [1] where a toxic gas is present, the cause of death could be due to poisoning, hypoxic asphyxia or a combination of both factors. In order to clarify the cause of death it is necessary to carry out three steps; (1) to determine the concentration of the gas in tissue samples, (2) to obtain data from animal experiments concerning the distribution of the gas in a body under various atmospheric concentrations of the gas and (3) to compare the two sets of data obtained with reference to other literature, if necessary.

Using these methods we have previously investigated the manner of death of victims in coal mines where methane gas was accidentally released from coal beds [2–4]. In the present paper we describe studies carried out using the same methodology to determine the cause of death of a victim who died in a tank where gaseous dimethyl sulfide had accumulated.

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Case report

In a paper manufacturing plant in Ebetsu, Hokkaido, Japan, a fatal accident occurred in a round storage tank (5 m in height, 4 m in diameter). Two men entered the tank to clean the bottom, where they immediately collapsed and were retrieved. One was already dead and the other died 1 1/2 days later. It was suspected that toxic gases had entered the tank through a pipe from another tank and had accumulated at the bottom. Later, the atmosphere was sampled to determine the concentrations of toxic gases; hydrogen sulfide was not detected, methyl mercaptan was present in a concentration less than 10 ppm, dimethyl sulfide (DMS) several ppm and dimethyl disulfide less than 1 ppm.

Autopsy. The 25-year-old man was already dead when retrieved and an autopsy was performed 27 h after the accident. A disagreeable odour smelling of garlic, was detected on the body surface and in the mouth. Lividity was tinted brown and petechial haemorrhages were seen in the conjunctivae. The cardiac blood also had a tint of brown and was fluid. The left lung weighed 600 g, the right 760 g and both were rich in blood and oedematous. Other organs were also congested. Blood and organ samples were taken for toxicological analyses. Microscopically, no abnormalities could be detected except for congestion in every organ.

Materials and methods

Identification of the inhaled gas. Blood was placed into a sealed vial, heated at 60°C for 30 min and the headspace gas phase was subjected to gas chromatography-mass spectrometry (GC-MS) (JEOL,

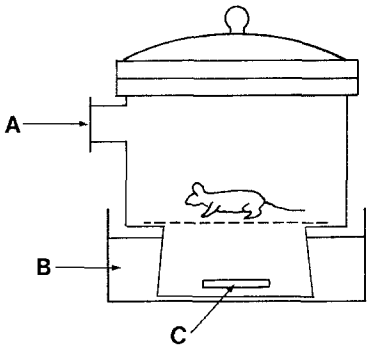

Fig. 1. Apparatus for experimental exposure of mice to gaseous dimethyl sulfide (DMS) in a confined space. (3 l-desiccator) *A*: adhesive vinyl tape; *B*: water bath; *C*: stirring bar

JNS-DX 300, equipped with a 1 m glass column packed with 10% SE-30). The temperatures of the injection port and the column were 280°C and 70°C, respectively. The carrier gas was helium (0.6 kg/cm^2). The ionization energy for electron impact mass spectra was 70 eV.

Quantification of DMS in tissues. DMS concentrations were determined by the headspace GC method as described elsewhere [5]. Briefly, 0.2 ml blood or 0.5 g tissue was put into a 10 ml vial, sealed using a piece of Teflon tape to avoid infiltration of the gas into the gum-cap and heated at 60°C for 4 h. The headspace gas (20 µl) was subjected to GC [6] using a gas chromatograph (GC-7AG, Shimadzu, Kyoto, Japan) equipped with a flame photometric detector and a 3 m glass column packed with 10% polyphenyl ether OS-124 on Shimalite TPA 60/80 mesh. The temperatures of the column and the detector were 60°C and 120°C, respectively. Pressure of air and hydrogen gas was adjusted to 1.2 and 0.7 kg/cm^2, respectively. The carrier gas was nitrogen (100 ml/min).

Animal experiments. Mice, weighing about 20 g, were exposed to an atmosphere containing gaseous DMS in a desiccator as illustrated in Fig. 1. Five different concentrations of DMS gas were investigated and 5 mice were used for each group. Liquid DMS was placed in the bottom of the desiccator which was immediately covered with a lid. The underside was warmed to 40°C in a water bath to vapourize the DMS and the gas mixed with air from the atmosphere with a stirring bar. As DMS was vapourized, the lid was raised slightly by the expanded atmosphere, some of which overflowed from the desiccator. After the overflow had ceased the lid was opened slightly, a mouse was inserted into the atmosphere and the lid was closed immediately. A small amount of the atmosphere was sampled to determine the concentration of DMS in the atmosphere by inserting a micro-syringe through the adhesive vinyl tape closing a small outlet at the side of the desiccator. The behaviour of the mouse after exposure to the atmosphere was observed and the time elapsed until changes in gait and respiratory arrest were also noted. After death had occurred, the atmosphere was sampled again to determine the concentration of DMS in the atmosphere. Blood and tissue samples were taken, weighed, sealed in glass vials and stored at −20°C until quantification of the DMS content was carried out.

This experiment was approved by the Animal Care and Use Committee, Hokkaido University School of Medicine, Japan.

Results and discussion

From the circumstances of the case it was thought that the death of victim had occurred over a very short period of time from poisoning by sulfur compound gases and/or from asphyxia due to the hypoxic atmosphere by substitution of the gases. The pathological findings were consistent with the above causes of death, which would have occurred immediately.

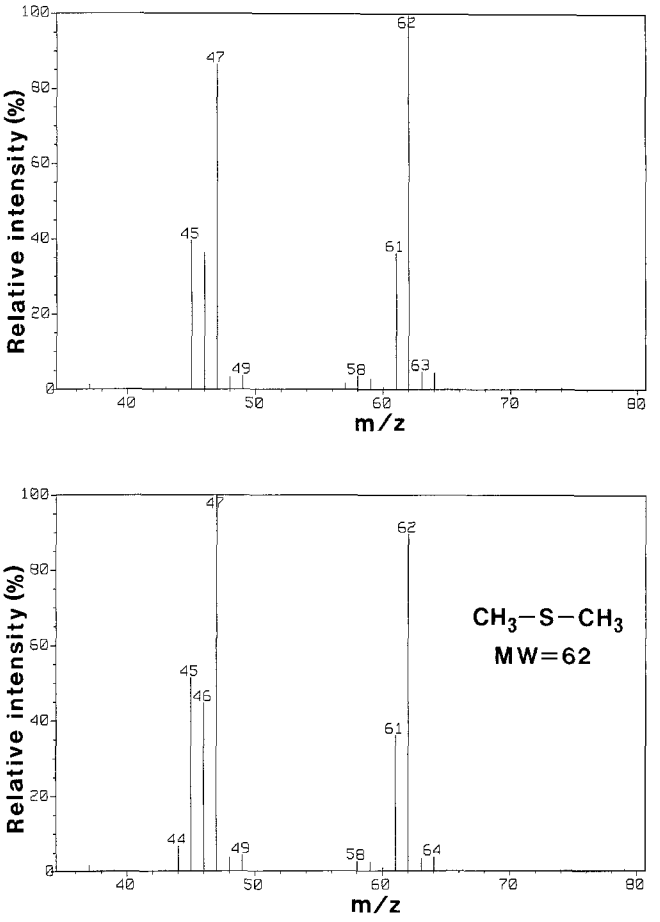

Fig. 2. Mass spectra of the inhaled gas recovered from the blood of the victim *(upper)* and authentic dimethyl sulfide (DMS, *lower*)

Table 1. Concentration of DMS in tissues of the victim

Tissue	DMS mg/g (wet weight)
Lung	0.11
Blood	0.20
Heart muscle	0.08
Brain	0.23
Liver	0.06
Spleen	0.21
Kidney	0.14
Muscle	0.01
Adipose tissue	0.04

The inhaled gas was identified by GC-MS as DMS, $(CH_3)_2S$. A single exogenous peak was observed by GC and the mass spectrum was in agreement with the control DMS as shown in Fig. 2.

The level of DMS in each tissue of the victim is listed in Table 1. DMS is insoluble in water but soluble in organic solvents such as ethyl alcohol and diethyl ether [7],

Table 2. Periods of time beginning of inability to stand (left hand column) and respiratory arrest (right hand column) after exposure of mice to an atmosphere containing various concentrations of dimethyl sulfide (DMS)

DMS (%)	Mice				
	#1	#2	#3	#4	#5
6.8 ± 1.3[a]	0.67 / 7.63[b]	0.77 / 3.42	0.77 / 4.33	0.80 / 7.17	1.08 / 7.03
11.6 ± 0.5	0.33 / 2.47	0.53 / 3.47	0.57 / 1.57	0.57 / 4.20	0.67 / 4.03
23.6 ± 3.7	0.33 / 1.08	0.35 / 0.80	0.35 / 1.27	0.37 / 2.35	0.47 / 1.47
34.0 ± 3.1	0.27 / 1.50	0.30 / 1.48	0.32 / 1.07	0.33 / 1.67	0.42 / 2.00
50.6 ± 3.6	0.13 / 0.77	0.17 / 0.73	0.18 / 0.67	0.20 / 0.72	0.23 / 1.17

[a] Mean ± S.D. ($n = 5$)

[b] Minutes

Table 3. Distribution of dimethyl sulfide (DMS) in tissues of mice exposed to an atmosphere containing DMS

Tissue	DMS (%)				
	6.8 ± 1.3	11.6 ± 0.5	23.6 ± 3.7	34.0 ± 3.1	50.6 ± 3.6
Lung	0.19 ± 0.21[a]	2.90 ± 1.57	1.66 ± 0.73	2.12 ± 0.44	0.29 ± 0.32
Blood	0.76 ± 0.16	5.34 ± 0.94	2.30 ± 0.38	4.54 ± 1.91	0.96 ± 0.29
Heart muscle	1.20 ± 0.72	3.96 ± 1.16	4.06 ± 1.36	3.78 ± 0.88	1.42 ± 0.36
Brain	1.16 ± 0.36	9.12 ± 2.77	8.22 ± 3.80	8.00 ± 2.92	0.88 ± 0.32
Liver	1.28 ± 0.74	4.50 ± 1.33	2.10 ± 0.97	2.24 ± 0.50	0.34 ± 0.14
Spleen	0.40 ± 0.18	2.20 ± 0.99	1.20 ± 0.47	1.48 ± 0.50	0.20 ± 0.09
Kidney	1.10 ± 0.34	3.62 ± 0.67	1.60 ± 1.08	1.94 ± 0.55	0.48 ± 0.13
Muscle	0.92 ± 0.55	1.52 ± 0.71	1.06 ± 0.26	2.14 ± 1.05	0.38 ± 0.10

[a] Mean ± S.D. (mg of DMS/g of wet weight) ($n = 5$)

which would indicate that DMS gas in a body might behave similarly to methane gas. A comparison of the levels of DMS in the victim with those of methane in rats killed by exposure to atmosphere containing only methane [4] indicated that the cause of death was probably asphyxia due to exposure to an anoxic atmosphere (namely DMS gas only) because in both cases, the concentrations (DMS or methane) in the lung tissue were not the highest when compared to other tissues and in adipose tissue was very low.

In order to diagnose the cause of death more accurately, animal experiments were carried out. The results are summarized in Tables 2 and 3. Five concentrations of DMS in atmospheric air were prepared: 6.8%, 11.6%, 23.6%, 34.0% and 50.6% (v/v). The DMS concentration in the atmosphere of the desiccator could be raised to a maximum of approximately 60%. A concentration of 100% DMS could not be reached because vapourization occurred at 40°C and DMS condensed on the inner wall of the desiccator (b.p. 36.2° or 38°C [7]). Every mouse died within about 8 min at a concentration of 6.8 ± 1.3% DMS. They became unable to move around 0.7–1 min after exposure. This is similar to previous observations on the acute toxicity and fatality of DMS inhalation as follows: exposure to 4% DMS for 4 h killed 50% of rats within 24 h [8]: a concentration of 5% DMS killed 1 rat in 15 min [9]: a concentration of 9.6% DMS caused 50% rats to be comatose [10]. As the DMS concentration increased, the time until the onset of inability to stand and respiratory arrest became shorter (Table 2). At 23.6 ± 3.7% and 34.0 ± 3.1% DMS no significant differences between the two periods could be seen. At a concentration of 50.6 ± 3.6%, mice became comatose in 8–14 s and died within about 1 min, which is equivalent to 2 periods of exposure to atmosphere without oxygen (100% methane) [4] and indicates that the cause of death was additionally caused by asphyxia due to the hypoxic atmosphere (about 10% oxygen).

DMS was distributed in all tissues of the mice at levels of 0.05–12.9 mg/g (data not shown) with an average of 2.34 mg/g (Table 3). At a concentration of 6.8 ± 1.3%, DMS was distributed in every tissue almost equally except for the lungs where the concentration was much lower. This situation has been named "type A". At concentrations of 11.6 ± 0.5%, 23.6 ± 3.7% and 34.0 ± 3.1% distribution patterns were almost the same. The DMS levels were generally higher than those in the type A and most highly accumulated in brain tissue. This pattern is named "type B". At a concentration of 50.6 ± 3.6%, DMS was distributed in lung, blood, heart muscle and brain at similar levels to those in the type A, but in the other tissues at lower levels than type A, this case is named "type C". A comparison of the types A, B and C showed that DMS accumulated more in type B and less in types A and C. It is thought that in type C, the inhaled DMS entered the blood through the alveolar walls and almost simultaneously reached the heart, but death occurred before the DMS had circulated sufficiently to raise it to the levels in other tissues. The mechanism of death from DMS may be due to the effect on the brain [10], but the brain levels in types A and C were much lower than those in type B. This suggests the possibility of different effects such as cardiac inhibition or primary shock. In type C, asphyxia due to hypoxic atmosphere may have

additionally contributed to the cause of death. The DMS blood level needed to cause coma by the inhalation of the gas has been reported to be 0.43 mg/ml [10], which is lower than the fatal DMS blood level obtained in our experiments (Table 3).

Comparing the DMS levels in the victim's tissues (Table 1) with those obtained from the animal experiments (Table 3), type B in mice does not agree with the results of our human victim, type A is found only in the lungs and spleens and type C is in closer agreement with the human case than type B except for the level in heart muscle. Rats are more resistant to hypoxic atmosphere [4] than humans who are reported to die immediately in a concentration of 5% oxygen [11], indicating the humans are more sensitive to hypoxia. Therefore it is thought that the victim would have died of a combination of DMS poisoning and asphyxia due to a lack of atmospheric oxygen (about 10%) substituted by the DMS gas. The gas would have been present at about 50% or more in the atmosphere of the tank at the time of the accident.

A fatal case of DMS poisoning has been reported after inhalation of methylmercaptan and DMS [12]. The lungs, blood, liver and kidneys contained both substances at about the same level. The tissue concentrations of DMS were 0.4–1.3, 17.9, 4.6–18.0 and 2.0–4.5 mg/g, respectively. This level is considered to be fatal, since it corresponds to the type B in our animal experiments.

In order to explain the manner and the cause of death of a victim in a confined space, it is important (1) to analyse the atmosphere as soon as possible after the accident [3, 13], (2) to determine the concentrations and distibution of the gas in the victim's tissues [2–4, 13] and (3) if necessary, to carry out animal experiments as demonstrated here and by Terazawa et al. [4].

References

1. Greenberg SR (1989) The confined space. A review of a recently discerned medical entity. Am J Forensic Med Pathol 10:31–36
2. Takatori T, Terazawa K (1980) A case report: determination of methane gas in cadaveric tissues from a coal-mine accident by gas chromatography. Hokkaido J Med Sci 55:363–365
3. Takatori T, Tomii S, Terazawa K (1981) Medicolegal studies on death from coal-mine accident by gas spurt (in Japanese). Jpn J Legal Med 35:462–467
4. Terazawa K, Takatori T, Tomii S, Nakano K (1985) Methane asphyxia coal mine accident investigation of distribution of gas. Am J Forensic Med Pathol 6:211–214
5. Terazawa K, Kaji H, Akabane H, Takatori T (1991) Determination of dimethyl sulphide in blood and adipose tissue by headspace gas analysis. J Chromatogr 565:453–456
6. Kaji H, Hisamura M, Saito N, Murao M (1978) Gas chromatographic determination of volatile sulfur compounds in the expired alveolar air in hepatopathic subjects. J Chromatogr 145:464–468
7. The Merck index, 11th edn (1989) Merck, Rahway
8. Tansy MF, Kendall FM, Fantasia J, Landin WE, Oberly R (1981) Acute and subchronic toxicity studies of rats exposed to vapors of methyl mercaptan and other reduced-sulfur compounds. J Toxicol Environ Health 8:71–88
9. Ljunggren G, Norberg B (1943) On the effect and toxicity of dimethyl sulfide, dimethyl disulfide and methyl mercaptan. Acta Physiol Scand 5:248–255
10. Zieve L, Doizaki WM, Zieve FJ (1974) Synergism between mercaptans and ammonia or fatty acids in the production of coma: a possible role for mercaptans in the pathogenesis of hepatic coma. J Lab Clin Med 83:16–28
11. Akaishi S (1967) Rinshoi no tame no hoigaku (in Japanese). Nankodo, Tokyo
12. Takahama K, Kikuda Y (1986) A fatal case of acute methylmercaptan poisoning (in Japanese). Res Pract Forensic Med 29:101–106
13. Manning T, Ziminski K, Hyman A, Figueroa G, Lukash L (1981) Methane deaths? Was it the cause? Am J Forensic Med Pathol 2:333–336