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Case report

Domino effect: An unusual case of six fatal hydrogen sulfide poisonings in quick succession

Nunziata Barbera, Angelo Montana *, Francesca Indorato, Nadia Arbouche, Guido Romano

Department "G.F. Ingrassia", Laboratory of Forensic Toxicology, University of Catania, Via S. Sofia, 87, Comparto 10, 95123 Catania, Italy

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ABSTRACT

Hydrogen sulfide (H_2S) is one of the most serious toxic gases encountered in forensic practice. Aside from being a by-product of many industrial processes, this gas is naturally produced during the putrefaction of organic substances. We report six autopsy cases of fatal H_2S poisonings from inhalation of H_2S gas after an occupational accident. These six men died during the unblocking of a wastewater cistern. The first worker died shortly after clearing the obstruction, the other five died, one by one, as they attempted to help their colleagues. The macroscopic and histological findings are discussed here to provide useful information for future cases. Greenish discoloration of the skin and of internal organs (liver, trachea, esophagus, stomach) was observed, and one case showed signs typical of drowning. We present a very unusual incident, complete with rare photographs and toxicological analysis. In these cases, based on both macroscopic and microscopic findings, the cause of death was most likely an inhibitory effect on cellular cytochrome oxidase causing respiratory failure.

1. Introduction

Hydrogen sulfide (H_2S) is one of the major toxic gases encountered in forensic practice [1]. It is a highly flammable colorless gas with a strong odor of rotten eggs. Aside from being a by-product of many industrial processes, this gas is naturally produced during the putrefaction of organic substances.

H_2S is also involved in moderating various physiological responses including anti-inflammation [2], reducing oxidative stress [3], neuromodulation [4], vasoregulation [5], protection from reperfusion injury after myocardial infarction [6] and inhibition of insulin resistance [7].

H_2S poisoning can occur following exposure to the gas, and the clinical symptoms depend on the concentration of the inhaled gas. At concentrations over 30 parts per million (ppm), the odor can be detected and the olfactory nerve may become paralyzed at levels above 150 ppm. Pulmonary and eye irritation occurs at exposure levels over 200 ppm. Coma and cardiopulmonary arrest occur at levels over 700 ppm, usually resulting from brain respiratory center paralysis, asphyxia, and cardiac failure. A very high H_2S concentration ($\geq$1000 ppm) immediately leads to unconsciousness and cardiopulmonary arrest; this is referred to as "knockdown".

Exposure for a few hours to concentrations of 500 ppm and above is likely to be fatal. Exposure for only a few minutes to concentrations of 1000 ppm and above is likely to cause rapid unconsciousness and death. At higher concentrations, death is caused by inhibition of brain respiratory centers; at lower concentrations, death is caused by pulmonary edema and congestion. Many fatalities have occurred in workers exposed to H_2S in confined spaces such as manholes [8].

We report the autopsy results for a very rare incident of six fatal occupational accidents involving H_2S poisoning where the men died in quick succession, in a lethal "domino effect". These six men were attempting to unblock a wastewater cistern when they died.

2. Case report

The first employee was attempting to unblock the valve of a sludge recycling tank in a wastewater treatment system. Immediately after removing the obstruction, a concentrated emission of H_2S gas occurred and he died very quickly. His colleague, who was in close proximity, died very soon after this. The third worker, seeing the scene from the top of the cistern, attempted to help them but became unconscious from the high concentration of H_2S and subsequently suffered cardiopulmonary arrest. By this stage, the sludge had started to flow out through the unblocked valve; two other employees were overwhelmed by the gas in the attempt to help their colleagues, and died one

* Corresponding author. Tel.: +39 95 378 21 64/32 876 55 428 (M); fax: +39 95 378 21 65.
E-mail address: angelomontana49@gmail.com (A. Montana).

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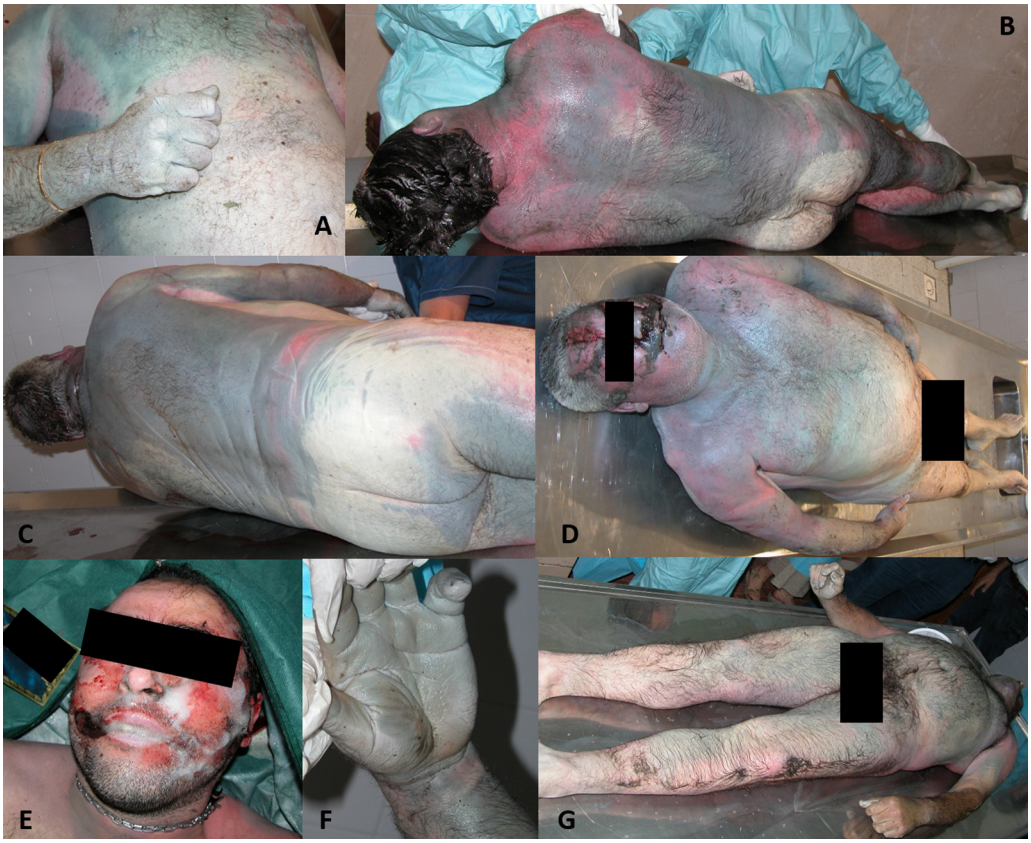

Fig. 1. External examination of the victims, showing greenish discoloration of the hypostasis over the whole body (A: 1st worker) (B: 2nd worker) (C, D: 3rd worker) (F: 4th worker) (G: 5th worker); extensive bleeding from the nose (D); foam at the mouth (drowning) and a few abrasions on the face of the 6th worker (E). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

after the other. In these two individuals, the high H_2S concentration immediately led to brain respiratory center paralysis, asphyxia and cardiac failure. The sixth worker lost consciousness and then died by drowning in the sludge.

Complete autopsies were performed approximately 36 h later. External examinations showed greenish discoloration from hypostasis over the whole body (Fig. 1). The autopsies revealed hyperinflated and overexpanded lungs and greenish discoloration of internal organs (Table 1; Fig. 2).

3. Materials and methods

At the six autopsies of this fatal H_2S poisoning case, femoral blood samples were collected. For the toxicological analysis, the detection and quantification of H_2S in blood was carried out using a 6890N Gas Chromatograph (Agilent HP, USA) coupled with an Agilent MSD 5973 mass selective detector.

3.1. Blood sample extraction

Blood aliquots (0.2 mL) were added to a mixture of 0.5 mL of 20 mM pentafluorobenzyl bromide PFB-Br solution in toluene, 2.0 mL of internal standard solution (10 mM of TBB in ethyl acetate) and 0.8 mL of 5 mM TDMBA solution in oxygen-free water saturated with sodium tetraborate. The preparation was vortexed for 1 min, and 0.1 g of potassium dihydrogen phosphate was added to the mixture. The preparation was again vortexed for 10 s and centrifuged at 2500 rpm for 10 min. An aliquot of the organic phase was then injected into the GC/MS apparatus.

4. Results

In the six fatal cases reported here, H_2S was detected in all femoral blood samples; the concentrations ranged from 8.7 to 28.6 mg/L (Table 2).

Table 1
Macroscopic and histological study results.

Cases	Sex	Age	Pathology findings
1st worker	M	45	Brain blue-green, skin red-brown, pulmonary edema
2nd worker	M	38	Few abrasions on the body, edema and congestion of brain, lungs, spleen and liver. Greenish discoloration of the internal organs
3rd worker	M	42	Cyanosis grayish-green, pulmonary edema. Cerebral cortex and nuclear masses greenish-purple and liver greenish
4th worker	M	39	Lung, liver and kidney greenish; glottis, trachea and bronchia revealed extensive erosion and submucosal hemorrhage
5th worker	M	51	Pulmonary edema with low protein content. Focal segmental glomerulosclerosis. Congestion present in brain, kidneys and spleen
6th worker	M	43	Externally, presence of foam at the mouth. Internally, diffuse congestion and petechial hemorrhage of the internal organs (meningeal congestion and cerebral edema); lungs are waterlogged, over-distended and heavier than expected. Pleural effusion, black fluid collected in the stomach, serious and hemorrhagic pulmonary edema with intra-alveolar edema and dilation of the alveolar spaces with secondary compression of the septal capillaries