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Case report

Fatal occupational inhalation of hydrogen sulfide

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Abstract

A young man aged 22 years, a sewer worker by profession, died after massive inhalation of hydrogen sulfide while at work. He was rescued by the emergency services and admitted to the critical care department, where he died due to massive myocardial necrosis less than 24 h after admission.

In this case, where the causes of the accident were not clearly established, autopsy and anatomopathologic examination made it possible to confirm the causal lesions which resulted in death and to question the initial version of the circumstances of the accident. Medicolegal investigation was valuable in determining possible liabilities and repercussions on coverage as an industrial accident by the national health insurance system.

Keywords: Forensic investigation; Toxicology; Hydrogen sulfide inhalation; Fatal intoxication

1. Introduction

Exposure to volatile substances is still a topic of current concern. These substances spontaneously or easily convert to a gaseous state and are particularly dangerous because of their depressive effect on the central nervous system. Hydrogen sulfide is one of the most common toxic gases. It is involved in a large number of industrial processes, either as raw material or as a waste product, but it may also be insidiously released by anaerobic bacterial decomposition of organic matter.

We report a case of rapidly fatal intoxication by hydrogen sulfide inhalation in a sewer worker, and demonstrate the value of medicolegal investigation in order to determine possible liability.

2. Case report

A man aged 22 years, employed with a sewage treatment company, was engaged in cleaning out sewer pipes. The work was carried out by a team of two men. They were required to wear special equipment, including a safety mask, as the main risk of this occupation lies in the emission of gases, such as cyanide, methane, carbon monoxide or hydrogen sulfide.

At 10:40 h the emergency services received a call from the colleague of the young man, who had accidentally fallen into a manhole while he was not wearing any protective equipment. The rescue team arrived 10 min later. They attempted to enter the manhole but were forced to give up the attempt at the entrance because of respiratory irritation, while the young man lay unconscious at a depth of about 5 m. Because of the narrowness of the manhole, the team were not able to enter it with their usual equipment and they set up a mechanical ventilation system to disperse the gas. The victim was brought to the surface half an hour later. He had a Glasgow coma scale score of 7 and convulsion crisis. Severe dyspnea required intubation and assisted ventilation; hemodynamic stability required norepinephrine perfusion.

With their wide band detection device (Drager® device type), the rescue team excluded a high concentration of cyanide, methane and carbon monoxide. The atmospheric measurement of hydrogen sulfide was only possible 1 h after the accident, using a device with an upper detection threshold of 30 parts per million (ppm). The reading, taken at less than 1 m below the surface after partial ventilation of the manhole, indicated that the concentration exceeded the upper threshold of the apparatus.

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Table 1
Results of blood tests carried out during hospitalisation of the victim

	First sample D1 (14:53 h)	Second sample D1 (21:38 h)	Third sample D2 (7:17 h)
Leukocytes (Giga/l)	14.32	12.05	15.67
Bicarbonates (mmol/l)	27.1	23.7	17.1
Creatinine (μmol/l)	113	97	105
Lactates (mmol/l)	1.72	1.12	1.86
Troponin (μmol/l)	0.63	7.66	11.76
CPK (IU/l)	253	644	6641
ASAT (IU/l)	30	40	185
ALAT (IU/l)	16	14	24
LDH (IU/l)	374	413	715
pH	7.27		7.33
PaO_2 (mmHg)	70		68
$PaCO_2$ (mmHg)	53		36

In intensive care unit (ICU), the victim presented bilateral myosis and regular tachycardia (heart rate 115 beats/min), hypotension at 108/88 mmHg (hemodynamic stability with pressor agent) and hyperthermia at 38 °C. Oxygen saturation was stable at 93%. A whole-body CT scan performed a few hours after the patient's admission confirmed the absence of body lesions and showed minor cerebral edema. The first blood tests were carried out at 14:53 h. Total CPK was elevated while the troponin level was unchanged (Table 1). Cardiac cytolysis was biologically confirmed at 21:38 h. ECG and ultrasound showed severe posterolateral hypokinesis of the cardiac muscle.

Given the atmospheric measurement, being almost certain about the diagnosis and considering the low interest of the therapeutic management, the measure blood concentration of hydrogen sulfide was not performed in ICU. Standard toxicological tests were negative; in particular, this confirmed the absence of volatile gases.

The victim died less than 24 h after admission to ICU, with acute respiratory distress complicated by severe hemodynamic insufficiency due to myocardial necrosis.

Autopsy confirmed that traumatic injury was restricted to a few abrasions. The brain was edematous and congested, and the heart showed gross changes and a generally heterogeneous appearance (Fig. 1).

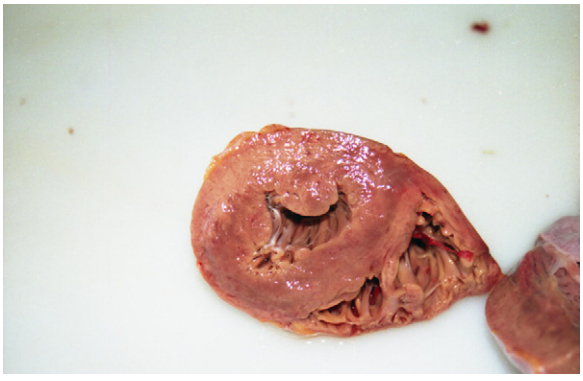

Fig. 1. Heterogeneous staining of the myocardium.

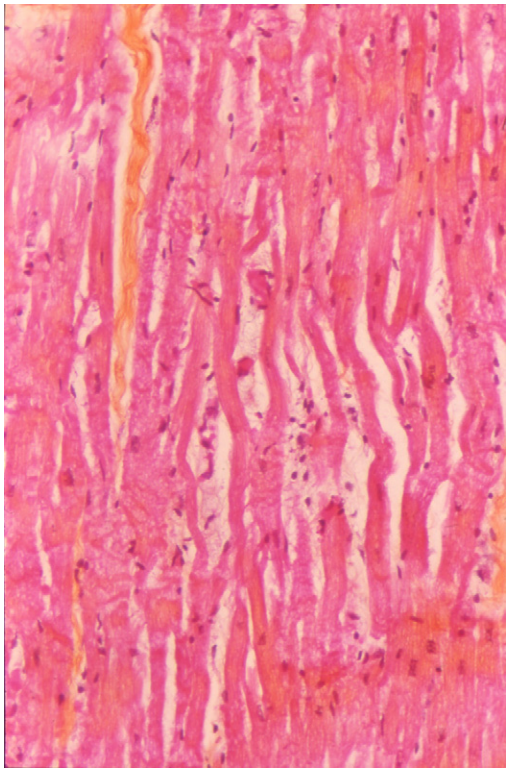

Fig. 2. Massive acidophilic myocyte necrosis.

Anatomopathologic examination confirmed the severity of ischemic and anoxic brain lesions, large areas of acidophilic necrosis without contraction bands (Fig. 2) with initial signs of recovery (some polymorphonuclear neutrophils along the connective trabeculae) and inhalation pneumonia. As cardiovascular arrest had not been observed during hospitalization, in this context the cardiac lesions were attributed to the toxic effects of hydrogen sulfide.

3. Discussion

3.1. Origin, toxicity and clinical effects of hydrogen sulfide

At room temperature and atmospheric pressure, hydrogen sulfide is a colorless, inflammable and explosive gas. It is heavier than air and therefore builds up in the lower levels of cavities, depressions or hollows. It is a component of all decomposing organic products through the action of sulfite-reducing bacteria. Hydrogen sulfide is one of the major causes of death by intoxication in the workplace and exposure to this gas is a risk in over 70 occupations [1]. It is given off by water treatment plants, during the cleaning out of sewers or descaling of pipes, in particular during treatment in an acid medium.

Inhalation is the main mode of human exposure, and the gas is then rapidly resorbed in the lungs. The unpleasant smell of rotten eggs is noticeable at 0.1 ppm. It is attenuated and becomes sickly sweet with increasing concentrations; above 100 ppm, olfactory saturation occurs and individuals can no longer detect the presence of this lethal gas. In our case, no particular odor was reported by the various persons present,