CASE REPORT

Fatal hydrogen sulphide poisoning in unconfined spaces

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Abstract

Fatal hydrogen sulphide poisoning usually occurs in confined spaces. We report two fatal accidents in unconfined spaces. The first accident caused the death of three workers who entered an unconfined room in a silo of sludge at the same time that a truck dumped several tons of sludge from water purification stations. The hydrogen sulphide that had accumulated inside the silo spilled out into the interior of the room due to a ‘splashing effect’ caused by the impact of the dumped sludge. The second accident occurred when the foreman of a wastewater treatment plant entered one of the substations to perform routine checks and suddenly lost consciousness. Although he was rapidly transferred to an intensive care unit, death occurred a few hours later. Hydrogen sulphide production was, in this case, due to an ‘embolism effect’ produced by the displacement of wastewater when the substation pumps were activated. We suggest ways in which accidents such as these caused by sudden release of hydrogen sulphide can be prevented.

Key words

Hydrogen sulphide poisoning; unconfined spaces; work-related death.

Introduction

Hydrogen sulphide (H₂S) is a gas that is easily produced when three conditions coincide: the presence of sulphates and sulphate-reducing bacteria, anaerobic conditions and temperatures >20°C. These conditions are very common during the summer in drains, latrines, septic tanks, sewers and deposits of dung and other decomposing organic matter.

The most common cases of H₂S intoxication occur when someone enters a confined space in which H₂S has accumulated over time due to lack of air exchange.

Severe or fatal hydrogen sulphide intoxications are very uncommon in locations which lack the characteristics of confined spaces [1,2]. We report two fatal accidents in unconfined spaces.

Case reports

The ‘first’ accident caused the death of three male workers aged between 19 and 28 years who entered an unconfined room containing a silo of sludge at the same time that a truck dumped several tons of sludge from water purification stations. The hopper for receiving the sludge (Figure 1) had a capacity of 120 tonnes and dimensions 5 m wide, 6.5 m long and 7.8 m high, with its mouth standing 35 cm above the load-level surface. The face is hidden except for unloading of sludge. Unloading lasted only a few minutes, with 27 tons of mud being abruptly dropped from a height of 2 m onto the 59 tons already in the hopper.

One worker was checking a pump mechanism, while the truck was dumping the sludge into the silo. As he was climbing the stairs to leave this area, he lost consciousness. Two fellow workers went to his aid but also lost consciousness. The hydrogen sulphide that had accumulated inside the silo had spilled out into the interior of the room due to a ‘splashing effect’ caused by the impact of the dumped sludge (Figure 1).

An environmental study was carried out 6 h after the accident in the area where the first worker had been working and detected 100 p.p.m. of hydrogen sulphide (threshold limit value-short term exposure limit American Conference of Industrial Hygienists: 15 p.p.m.). Blood levels of sulphide of the three victims were 2.48, 14.9 and 18.1 mg/l, respectively, in autopsies carried out 24 h after the accident.

The second accident occurred when the 34-year-old male foreman of a wastewater treatment plant was carrying out a routine check of a wastewater pumping substation before its opening. As he descended the stairs, he suddenly lost consciousness. He was rescued ~15 min later by the emergency services (Figure 2) and transferred to...
a community hospital, where he remained for \( \sim 8 \) h in a coma. On the family’s wishes, the patient was transferred to another hospital located 400 km away but was dead on arrival. No test for sulphides or thiosulphate was performed. The diagnosis was made indirectly through identification of blackening of metal objects provided by the rescue team.

Hydrogen sulphide production was due to an ‘embolism effect’ produced by the displacement of wastewater when the substation pumps were turned on.

Discussion

Hydrogen sulphide poisoning can occur in situations other than in confined spaces. Our two cases occurred in unconfined spaces where there was natural ventilation for air renewal (as workers were often working in these areas) and easy access with stairs and railings.

Hydrogen sulphide acts on different body systems, including enzymes and metabolic pathways. It can cause local effects (irritative action), acting mainly on the mucous membranes of the airways, lungs, eyes and digestive organs. It is a central nervous system depressant, particularly of the respiratory centre, and inhibits cytochrome oxidase, preventing mitochondrial oxygen utilization and thereby blocking cellular respiration. Cells are forced to use a pathway anaerobically, with corresponding lactic acidosis [3]. The relationship between environmental levels of hydrogen sulphide and major health effects are shown in Table 1.

Patients who survive acute hydrogen sulphide poisoning may suffer post-anoxic neurological sequelae [4–6]. The diagnosis is based on known sources of pollution or investigation of the accident site for blackened metals, a characteristic of hydrogen sulphide, blood levels of hydrogen sulphide consistent with clinical intoxication [7–9], environmental levels of hydrogen sulphide (Table 1), blood levels of sulphide and blood and urine levels of thiosulphates [2,10]. McAnalley et al. [7] reported that blood levels of hydrogen sulphide in 100 patients were <0.05 mg/l and no >0.4 mg/l in 25 autopsy cases, including some which were badly decomposed. In our cases, blood levels were between 2.4 and 18.1 mg/l.

In Case 1, the environmental values obtained 6 h after the accident were similar to those described by Kuge et al. [10].

When intoxication occurs, those affected should be extracted from the toxic environment, but rescue
should never be attempted without self-contained breathing systems. Therapy should include 100% oxygen and ensuring correct metabolic acidosis with sodium bicarbonate, with cardiopulmonary resuscitation if needed. There is no specific antidote for hydrogen sulphide poisoning.

We suggest that the best ways to prevent accidents such as these produced by sudden release of hydrogen sulphide are as follows:

- Planning to ensure that actions that can generate potentially dangerous emissions are carried out in the absence of staff.
- Install powered gas extractors linked directly to the exterior of the building for potentially dangerous emissions.
- Install gas detection systems connected directly to high-volume ventilation systems that automatically trigger alarms. Manual systems may be slower to initiate ventilation and may be ineffective.

**Conflicts of interest**

None declared.

**References**


**Table 1. Effects of H₂S on humans**

<table>
<thead>
<tr>
<th>Hydrogen sulphide levels (p.p.m.)</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.003–0.02</td>
<td>Odour threshold</td>
</tr>
<tr>
<td>50</td>
<td>Eye and respiratory irritation</td>
</tr>
<tr>
<td>150</td>
<td>Olfactory nerve paralysis</td>
</tr>
<tr>
<td>250</td>
<td>Exposure may cause pulmonary oedema</td>
</tr>
<tr>
<td>500</td>
<td>Anxiety, headache, ataxia, dizziness, stimulation of respiration, amnesia, unconsciousness</td>
</tr>
<tr>
<td>750</td>
<td>Quickly unconscious; death without rescue</td>
</tr>
<tr>
<td>1000</td>
<td>Rapid collapse; respiratory paralysis leading to death</td>
</tr>
<tr>
<td>5000</td>
<td>Immediate death</td>
</tr>
</tbody>
</table>

*Adapted from Fuller and Suruda [1] and other sources [3,6].