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CASE REPORT

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# Fatal sequelae of hydrogen sulphide poisoning

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**Abstract**

Hydrogen sulphide ($H_2S$) is a toxic, colourless gas abundantly present at waste plants and sewers due to the presence of anaerobic forming organisms. Hazardous exposure via accidental, intentional or occupational contact results in endothelium disruption, cellular instability, decreased respiratory functional capacity and cardiovascular compromise with a rapidly fatal clinical course. Clinical manifestations are variable depending on the level of exposure with moderate or heavy exposure associated with rapid fatality. Respiratory manifestations remain the primary reason for admission to critical care facilities. We describe a case of a 30-year-old sewer worker with a history of heavy accidental occupational exposure of inhaled $H_2S$ admitted with acute respiratory distress syndrome and a rapid respiratory decline ultimately leading to death.

**KEYWORDS**

acute respiratory distress syndrome, hydrogen sulphide, poisoning

## INTRODUCTION

Hydrogen sulphide (H2S) is a toxic, colourless gas abundantly present at waste plants and sewers due to the presence of anaerobic-forming organisms. Hazardous exposure via accidental, intentional or occupational contact results in endothelium disruption, cellular instability, decreased respiratory functional capacity and cardiovascular compromise with a rapidly fatal clinical course. Clinical manifestations are variable depending on the level of exposure with moderate or heavy exposure associated with rapid fatality. Respiratory manifestations remain the primary reason for admission to critical care facilities.

## CASE REPORT

A 30-year-old sewer worker was referred to the intensive care unit (ICU) with severe acute respiratory distress syndrome (ARDS) secondary to submersion in faecal contaminated water. The history provided from the referring hospital was that the patient had fallen through a manhole into the underlying sewer system. On arrival at the ICU, he required significant mechanical ventilatory support with Pressure Synchronized Intermittent Mandatory Ventilation (PSIMV) with high inspiratory pressures, positive end-expiratory pressure (PEEP) and a fraction of inspired oxygen ($FiO_2$) of 0.7 to maintain an arterial saturation of 88%. The ratio of arterial oxygen partial pressure to the fractional inspired partial pressure of oxygen ($PaO_2/FiO_2$) was 98. He was shocked, requiring high doses of adrenaline at 4 mcg/kg/minute to maintain an adequate perfusion pressure. Bilateral diffuse crackles were evident on respiratory examination. The initial arterial blood gas showed hypoxaemia and a mild compensated metabolic acidosis (pH 7.35), with a $PaO_2$ of 9.20 kPa. He had a markedly low white cell count of $0.62 \times 10^9$/L (normal: 4–10 $\times 10^9$/L) and neutrophil count of $0.32 \times 10^9$/L (normal 2.00–7.00 $\times 10^9$/L), a significantly elevated C-reactive protein (CRP) of 199 mg/L (normal <5 mg/L) and a serum pro-calcitonin level (PCT) of 207.36 μg/L (normal: <0.5 ng/mL). He tested negative for Human Immunodeficiency Virus. The initial chest radiograph was consistent with acute respiratory distress syndrome (ARDS, Figure 1).

An assessment of severe ARDS secondary to submersion in contaminated water and resultant septic shock was made. Empiric broad-spectrum antibiotics (Meropenem and Vancomycin) were initiated, together with general supportive critical care, including haemodynamic resuscitation and lung protective ventilation.

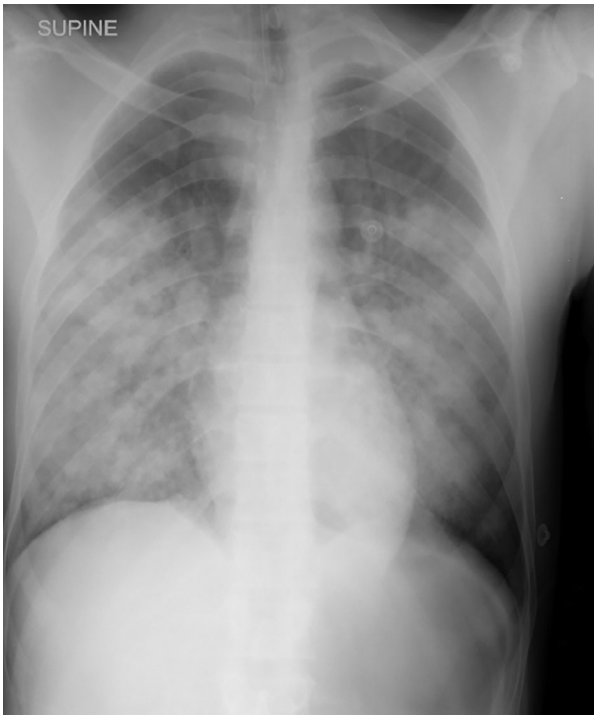


**FIGURE 1** The chest radiograph taken on admission showing diffuse alveolar infiltrates in keeping with non-cardiogenic pulmonary oedema / acute respiratory distress syndrome. (admission)

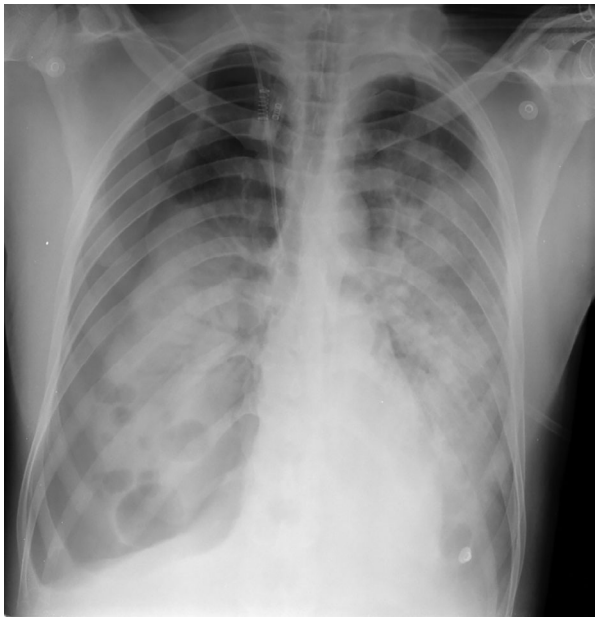

**FIGURE 2** This chest radiograph was taken minutes prior to the patient's death (1 day post admission). Note the right-sided pneumothorax. An intercostal drain was inserted

The patient continued to deteriorate with an increasing need for both haemodynamic and ventilatory support. Although extracorporeal membrane oxygenation (ECMO) was a consideration, in view of the rapidly increasing need for vasopressor support, he was deemed too unstable for transfer to the dedicated ECMO centre. Three days after admission, collateral history from the patient's colleague, a witness to the event, was obtained and suggested the strong possibility of inhalational exposure and lung injury. Contrary to the initial report, there was, in fact, no submersion in water but rather, a successful attempt made by the patient to pull a colleague out of the sewer through the manhole. Neither had been wearing their protective face mask at the time. Both had described the rapid onset of dyspnoea before requiring ventilation. The colleague, unfortunately, demised soon after admission to emergency unit of the hospital.

At the time of obtaining this vital history, the patient acutely deteriorated with life-threatening haemoptysis, worsening shock and ultimately cardiorespiratory arrest. T. Clinical assessment during the resuscitation efforts suggested a right-sided pneumothorax (Figure 2) in addition, and an intercostal drain (ICD) was promptly inserted. Pink frothy, foul-smelling (similar to rotten eggs) fluid was noted in the ICD. Unfortunately, following a very brief period of clinical improvement and return of spontaneous circulation after the insertion of the ICD, the patient experienced a second cardiac arrest and despite further resuscitative efforts, died. Microbiological reports known after the patient's death confirmed the lack of organisms isolated from the blood cultures or tracheal aspirate taken on ICU admission.

Given the circumstances, the case was referred for a forensic review and the cause of death was ultimately ruled as 'accidental $H_2S$ poisoning' by the attending forensic pathologist based on the findings of fibro-cavitary lung changes as well as elevated levels thiosulphate in lung tissue. This resulted in an official occupational hazard investigation by the relevant authorities.

## DISCUSSION

$H_2S$ is produced in areas such as sewers and industrial waste areas. In large quantities, it is inevitably lethal.[1] Local effects may involve the skin or eyes with erythema or conjunctivitis whereas generalized effects include, predominantly, cardiovascular, and respiratory instability.[1,2] Due to its similar mechanism of action to cyanide, it is occasionally used in suicide and often diagnosed at autopsy in developed countries.[3]

$H_2S$ inhibits mitochondrial function, resulting in aerobic metabolic arrest. As a result, markedly varied systemic effects are reported.[4] Although $H_2S$ poisoning predominantly occurs by inhalation, bloodstream inoculation can occur. Irrespective of the mechanism of entry, high-level exposures of $H_2S$ are very rapidly lethal.[4] In all documented cases, toxic respiratory manifestations remain the primary admission presentation to a critical care facility.[5] Respiratory compromise, due to decreased surfactant and surface tension disruption, leads to a fulminant pulmonary haemorrhage syndrome and rapid onset of fibroblast deposition.[6] This is almost inevitable after a high level of exposure.[7] Cardiovascular and endothelial

destabilization ensue, with resultant hypotension and possibly, cardiorespiratory arrest.[8]

Published case reports of fatality present with similar histories globally. Early recognition of exposure will prompt adequate directed management.[9] Establishing an accurate occupational history and detailing the extent of the exposure allows early referral to a critical care unit, especially if moderate or heavy exposure is suspected.[10] Typical chest radiograph patterns demonstrate pulmonary oedema in the acute phase alongside ground glass attenuation, which may develop at any stage of the insult.[11–13] Hyperbaric oxygen to acutely slow disease progression remains controversial, as its use, similarly, has not been reported to improve patient outcomes.[12] Sodium Nitrite or Cobamamide are suggested treatment options, converting $H_2S$ to the less toxic sulfmethaemoglobin.[5] However, these adjunctive measures have not been proven to decrease mortality. Evidence of survival after moderate or high levels of exposure is scant. In those that do survive, destructive lung parenchymal changes have been reported, occurring at variable rates depending on the setting.[5,6,13] Confirmation of $H_2S$ poisoning relies on the demonstration of its two main metabolites, thiosulphate or sulphate, in tissue or serum within 7 days. This may prove challenging as oxidative metabolism of the gas tends to occur rapidly prior to death and, post-mortem, putrefaction may affect the accuracy of diagnosis.[14]

In conclusion, this case highlights the rapid progression of inhalational $H_2S$ toxicity and fatality of inhalation without timeous intervention. Acute $H_2S$ poisoning should be considered in patients with rapidly progressive ARDS if occupational exposure to $H_2S$ is a possibility. Thorough, accurate history taking is crucial and rapid initiation of directed management should be instituted to potentially reduce fatal progression.[5,13–15]

## AUTHOR CONTRIBUTIONS

All authors were involved in the clinical management of the patient. NG and UL wrote the manuscript, that was critically reviewed by all co-authors.

## CONFLICT OF INTEREST STATEMENT

Coenraad F. N. Koegelenberg is an Editorial Board member of Respirology Case Reports and a co-author of this article. They were excluded from all editorial decision-making related to the acceptance of this article for publication. Coenraad F. N. Koegelenberg is an Associate Editor of Respirology Case Reports, the other authors have no conflict of interest to declare.

## DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

## ETHICS STATEMENT

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

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