Journal Homepage: -www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI:10.21474/IJAR01/23951
DOI URL: http://dx.doi.org/10.21474/IJAR01/23951

RESEARCH ARTICLE

ACUTE OCCUPATIONAL HYDROGEN SULFIDE POISONING IN A CONFINED SPACE: REPORT OF TWO CASES

ArmelB , El Rhabali G, Benabbdellah I, Belhacene A, Zarouali H, Sebbata O and El Moussaoui R

Manuscript Info

Manuscript History
Received: 8 June 2026
Final Accepted: 10 July 2026
Published: August 2026

Key words:-
hydrogen sulfide; occupational poisoning; respiratory distress; altered consciousness

Abstract

Hydrogen sulfide (H2S) is a highly toxic gas commonly encountered in sanitation systems and certain industrial settings [1,2]. Acute poisoning occurs mainly in occupational settings and constitutes a medical emergency due to the rapid onset of neurological and respiratory complications, whose severity depends on gas concentration and exposure duration [3,5]. We report two cases of accidental H2S poisoning in confined spaces among sanitation workers. Both patients were admitted to the emergency department with altered consciousness and respiratory distress, requiring endotracheal intubation and mechanical ventilation. Chest imaging revealed bilateral pulmonary lesions compatible with toxic lung injury. Both patients experienced favorable outcomes under supportive treatment. Diagnosis is based primarily on occupational exposure history and clinical presentation, complemented by laboratory and imaging studies to assess respiratory involvement and gas exchange impairment [6,7]. Management is mainly supportive,including rapid removal from the toxic environment, early oxygen therapy, and ventilatory support. The use of specific antidotes remains controversial, and their availability is limited [9]. These observations underscore the importance of preventing occupation al exposure to H2S, early recognition of this potentially lethal poisonin g, and prompt initiation of appropriate management to improve prognosis [11].

Introduction:-

Hydrogen sulfide (H2S) is a colorless gas with a characteristic “rotten-egg” odor, produced by the decomposition of organic matter. It is commonly encountered in sewage systems, wells, treatment plants, and certain chemical industries [1,4]. Exposure to H2S constitutes a major occupational hazard, especially in confined spaces, where rapid accumulation of the gas can lead to severe, sometimes fatal, poisoning [11]. H2S toxicity involves two main mechanisms: direct irritant effects on the respiratory mucosa, leading to acute lung injury and potentially non-cardiogenic pulmonary edema [7], and inhibition of mitochondrial cytochrome-c oxidase, resulting in histotoxic hypoxia, similar to cyanide poisoning [5,13,14]. Severity depends on gas concentration and exposure duration, with higher concentrations potentially causing olfactory paralysis, loss of consciousness, respiratory arrest, and cardiopulmonary arrest. Despite high lethality, reported cases are rare, likely due to underreporting and prehospital mortality [2,3]. We report two cases of acute occupational H2S poisoning in a confined space. Both patients were

admitted with acute altered consciousness and severe respiratory distress, requiring immediate management including oxygen therapy, airway protection, and mechanical ventilation.

Case Reports:-

Two men aged 34 and 42 years, with no significant medical history, were admitted to the emergency department for acute occupational hydrogen sulfide (H2S) poisoning. Exposure occurred during a sanitation intervention in a poorly ventilated sewer, without gas detection equipment. Patient 2 was initially exposed after remaining inside the sewer for several minutes. Patient 1 entered later to assist him and also fell into the sewer, losing consciousness immediately. Both patients were found unconscious by colleagues a few minutes after the incident. Exact exposure duration could not be determined.

Case 1:-

On admission, the patient had altered consciousness with a Glasgow Coma Scale (GCS) score of 10 (E4, V1, M5), without focal neurological deficit or clinical signs of seizure. Pupils were equal and reactive. Vital signs were stable: blood pressure 120/70 mmHg, heart rate 90, respiratory rate 22/min, and body temperature 35°C. Oxygen saturation was 99% on room air. Lung auscultation revealed coarse rales. Arterial blood gas showed moderate hypoxemia (PaO_2 76 mmHg). Laboratory tests revealed moderate hepatic enzyme elevation (AST ×3 N, ALT ×2 N, ALP ×2 N, γ-GT ×5 N), with normal renal function and blood count. Brain CT was normal. Chest CT showed bilateral pulmonary lesions compatible with toxic lung injury (Figure 1). Due to altered consciousness, endotracheal intubation was performed, followed by mechanical ventilation in the ICU. Clinical course was rapidly favorable, allowing extubation after 48 hours. The patient was discharged from the ICU on day 3.

Case 2:-

On admission, the patient had altered consciousness with GCS 9 (E3, V1, M5), associated with clinical signs of seizure, including tongue biting. Pupils were equal and reactive. Vital signs: blood pressure 140/80 mmHg, heart rate 80 bpm, respiratory rate 30/min, and temperature 36°C. Oxygen saturation was 88% on room air, corrected to 99% with 5 L/min oxygen. Lung auscultation revealed bilateral coarse rales (Table 1). Arterial blood gas showed severe hypoxemia (PaO_2 60 mmHg). Laboratory tests revealed hepatic enzyme disturbances similar to Case 1. Brain imaging was normal. Chest CT revealed bilateral lesions consistent with acute toxic pneumonitis. Endotracheal intubation and mechanical ventilation were performed due to respiratory distress and altered consciousness. Clinical course showed progressive respiratory improvement, allowing extubation on day 5. Total hospital stay was 12 days.

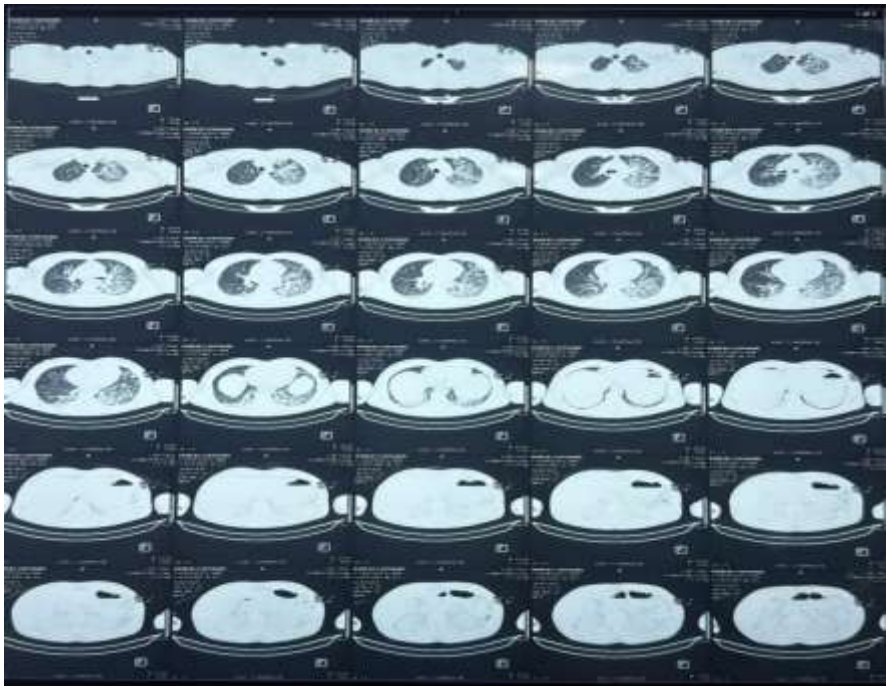

Figure 1: Chest CT of case 1

Table 1. Comparative clinical data of the two patients

Éléments	Cas n°1	Cas n°2
Age / Sex	34 years / Male	42 years / Male
Exposure order	Secondary (rescue)	Initial
Exposure environment	Sewer (confinedspace)	Sewer (confinedspace)
Initial neurological status	Glasgow 10, no seizure	Glasgow 9, seizure
Respiratory involvement	Moderatehypoximia	Severehypoxemia
Initial PaO_2	76 mmHg	60 mmHg
Chest CT	Bilateraltoxiclesions	Bilateraltoxiclesions
Hepatic involvement	Moderate	Moderate
Mechanical ventilation	Yes	Yes
Duration of ventilation	48 hours	5 days
Length of hospitalstay	3 days	12 days
Outcome	Rapidly favorable	Progressive favorable

Discussion: -

Acute H2S poisoning is a rare but potentially fatal toxicological emergency, most commonly occurring in occupational settings, particularly in confined spaces [1,2,11]. These cases illustrate the sudden onset of both neurological and respiratory symptoms typical of high-concentration exposure [3,5]. Both patients developed hypoxemia requiring invasive ventilation, with chest CT confirming bilateral acute toxic pneumonitis. This lung injury results from direct irritant effects of H2S on the respiratory epithelium combined with oxidative stress mechanisms leading to non-cardiogenic pulmonary edema [6,7]. The severity of respiratory involvement, especially in the second patient, explains the prolonged duration of mechanical ventilation and hospital stay [2,8]. Neurological manifestations, though present, were secondary to respiratory compromise. Altered consciousness and seizures were due to acute cerebral hypoxia from cytochrome-c oxidase inhibition by H2S [5,13,14]. Brain imaging was initially normal, consistent with literature reports, though subtle cerebral injuries and late neurocognitive sequelae have been described after severe exposure [10,12].

Both patients had transient moderate hepatic enzyme elevations, reflecting systemic H2S toxicity. These enzyme changes likely indicate mitochondrial and cellular injury, which may often be underestimated in acute poisoning but warrant monitoring [5,13]. Management relies on supportive care. Rapid removal from the toxic environment, early oxygen therapy, and mechanical ventilation are the cornerstones [4,11]. No antidotes were administered, highlighting that early symptomatic management remains the key determinant of favorable outcome [9]. These cases emphasize that the severity of respiratory involvement largely determines short-term prognosis in acute H2S poisoning. They also underscore the importance of preventive measures in occupational settings, including strict adherence to safety procedures and the use of gas detectors in confined spaces [4,11].

Conclusion: -

Acute occupational H2S poisoning is a life-threatening emergency, dominated by severe respiratory and neurological involvement [1,5]. Rapid extraction from the confined environment and early supportive care, including mechanical ventilation, are crucial to improving outcomes [6,11]. Early chest CT facilitates assessment of pulmonary injury and guides respiratory management [7]. Although transient, hepatic involvement reflects systemic toxicity [5,13]. These cases reaffirm the critical importance of preventive measures, including strict adherence to safety protocols and use of gas detectors in confined spaces [4,11].

References:-

1. Querellou E, Jaffrelot M, Savary D, Savry C, Perfus JP. Intoxication accidentellemortelle par hydrogènesulfuré [Fatal accidentalhydrogensulfidepoisoning]. Ann FrAnesthRéanim. 2005;24(10):1302–1304. doi:10.1016/j.annfar.2005.04.024.
2. Millot E, Compagnon F, Bouvet F. Les intoxications à l'hydrogènesulfuré : à propos de deuxcas [Hydrogensulfidepoisoning: report of two cases]. UrgencesMédicales. 1995;14(1):29–35. doi:10.1016/0923-2524(96)80608-4.
3. Chaari A, Bahloul M, Chelly H, Sahnoun M, Bouaziz M. Défaillancecardiaque et neurologiquesecondaire à une intoxication accidentelle par l'hydrogènesulfuré : à propos d'un cas [Cardiac and neurologicalfailuresecondary to accidentalhydrogensulfidepoisoning: a case report]. Ann FrAnesthRéanim. 2010;29(4):304–307. doi:10.1016/S0750-7658(10)00045-6.
4. Institut National de Rechercheet de Sécurité (INRS). Fiche toxicologique n°32 :Sulfured'hydrogène (H_2S) [Toxicological sheet No. 32: Hydrogensulfide (H_2S)] [Internet]. Février 1997 [cité 7 sept 2025]. Disponiblesur : https://www.inrs.fr/dam/ficheTox/FicheFicheTox/FICHETOX_32-1
5. Guidotti TL. Hydrogensulphide: advances in understandinghumantoxicity. ClinToxicol (Phila). 2010;48(2):93–106.
6. Reiffenstein RJ, Hulbert WC, Roth SH. Toxicology of hydrogensulfide. AnnuRevPharmacolToxicol. 1992;32:109–134.
7. Chaturvedi AK, et al. Acute respiratorytoxicity of hydrogensulfide. J ApplToxicol. 2013;33(6):453–460.
8. Lindenmann J, et al. Acute hydrogensulfide intoxication and systemiceffects. Occup Med (Lond). 2010;60(2):164–166.
9. Brenner M, et al. Cyanide and hydrogensulfide antidotes: controversies and updates. ToxicolRev. 2016;35(2):71–82.
10. Washtock J. Long-termneurologicaleffectsafterhydrogensulfideexposure. Neurotoxicology. 2009;30(6):1065–1070.
11. OccupationalSafety and Health Administration (OSHA). HydrogenSulfideHazards and Controls in ConfinedSpaces. U.S. Department of Labor.
12. Rumbeiha WK, Kim DS. Neurologicalsequelae of acute hydrogensulfidepoisoning: a literaturereview, controversies, and knowledge gaps. Neurol Int. 2025;17(5):71.
13. Cooper AJL, Brown GC. Impact of hydrogensulfide on mitochondrial and bacterialbioenergetics. Int J Mol Sci. 2021;22(23):12688.
14. Health Council of the Netherlands. Hydrogensulphide: mechanism of toxicity. Advisory Report.