Case Report

Sewer gas poisoning causing transient and focal ST-segment elevation in the ECG: Case report

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Abstract

In India, a large number of sanitary care workers are involved in manual scavenging. This exposes them to sewer gas mainly consisting of hydrogen sulphide. Sewer gas toxicity primarily causes neurological injury, followed by cardiac and respiratory involvement. A few cases of diffuse ST-segment elevation in the electrocardiogram (ECG) following hydrogen sulphide poisoning are known in the literature. Here, we report a case of acute sewer gas poisoning in a 45-year-old man with transient and focal ST-segment elevation in the anteroseptal leads of the ECG mimicking acute anterior wall myocardial infarction.

Keywords: ECG, hydrogen sulphide, sewer gas toxicity, TROP -T

Introduction

Sewer gas is a mixture of gases formed by the decomposition of wastes dumped in sewers from both households and industries. It mainly consists of hydrogen sulphide, methane, carbon monoxide, sulphur dioxide, and various nitrogenous gases.[1,2] Because of the closed nature of the underground sewer pipelines, these gases build up to very high concentrations and produce levels that are highly toxic to the human body. The main component is hydrogen sulphide, a colourless gas with the odour of rotten eggs. H_2S is directly toxic and causes cellular hypoxia by inhibiting mitochondrial cytochrome oxidase, which may rapidly lead to multi-organic dysfunction and death.[3] It can cause unconsciousness within seconds and hence also known as knockdown gas.[4,5] In the case of low levels of exposure (0.05 ppm), early signs and symptoms include the smell of rotten eggs, fatigue, headaches, nausea or vomiting, dizziness or lightheadedness, and poor memory and concentration. High levels of sewer gas exposure which occasionally occurs during manual scavenging leads to loss of smell, throat and eye irritation and congestion in eyes, mental depression (>500 ppm), seizures, coma, and even death immediately (>1000 ppm).

Case Report

Two sanitary care workers entered a sewer for manual scavenging. They became unconscious on entering the 15-feet deep sewer and were pulled out by their co-workers. The 24-year-old male died on the spot, whereas the other person, a 45-year-old male, was taken to a local healthcare facility. His Glasgow coma scale (GCS) was 5, his blood pressure (BP) was 150/80, and his electrocardiogram (ECG) was normal on arrival [Figure 1a]. He was electively intubated for poor GCS and referred to our hospital where he was admitted to the intensive care unit (ICU). On examination, the patient was unconscious, with a BP of 60/40 mm of Hg. His heart rate was 128/min, and chest

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Received: 08-03-2022 **Revised:** 17-05-2022
Accepted: 27-05-2022 **Published:** 31-10-2022

Access this article online

Quick Response Code:

Website: www.jfmpc.com

DOI: 10.4103/jfmpc.jfmpc_552_22

How to cite this article: Kumar L, Mawari G, Kumar N, Sarkar S, Daga MK. Sewer gas poisoning causing transient and focal ST-segment elevation in the ECG: Case report. J Family Med Prim Care 2022;11:6533-6.

auscultation revealed diffuse fine crepitations throughout the chest, suggesting alveolar oedema. His saturation by pulse oximetry was 82%. The central venous pressure of the patient was 8 cm of water column, and the inferior vena cava was 17.2 mm in size. The ECG of the patient showed ST-segment elevation in the anteroseptal leads V1 to V3 [Figure 1b]. His TROP T by the kit method came out to be positive, CPK was 338 u/L, Pro BNP was 2600 ng/ml, and D-dimer was 1059 ng/ml. The arterial blood gas of the patient revealed a PaO2/fiO2 ratio of 241; the pH was 7.285 with a HCO^{3-} level of 16.9 mmol/L, suggesting acidosis. Bedside 2DEcho was performed. It showed anterior wall hypokinesia with an ejection fraction of 35%. A diagnosis of acute sewer gas poisoning with heart failure and acute respiratory distress syndrome (ARDS) was made. The patient was managed conservatively and put on the assist/control mode of ventilation. The initial ventilator settings were fiO2 100% with a positive end-expiratory pressure of 6, a respiratory rate of 14/min, and a tidal volume of 5 ml/kg. Antibiotic therapy was initiated and fluids were given to maintain his mean arterial pressure >65 mm of Hg along with ionotropic support of nor-adrenaline (1 microgram/kg/min) and low-dose dobutamine (1 microgram/kg/min). Hydrocortisone was given as a 200 mg stat dose, followed by 100 mg i.v. three times a day (TDS) for ARDS. Prophylaxis for deep venous thrombosis was initiated with low-molecular weight heparin. The patient CPK level increased in the next 2 days and reached up to 1593 u/l. The patient showed signs of improvement regaining consciousness and maintaining BP, and hence, inotropes were tapered off. Bedside 2DEcho was repeated, and it showed improvement in ejection fraction going up to 40%. On day 4, he was successfully extubated and kept under watchful observation. He did not give consent for coronary angiogram initially and hence managed conservatively. His CPK levels came down to 600 u/L. The repeat bedside 2DEcho showed no regional wall motion abnormalities and a left ventricular ejection fraction of 55%. The ST elevation in the ECG also returned to the baseline [Figure 2]. Post recovery from ARDS, a chest x-ray was performed, in which mild interstitial opacities were observed [Figure 3]. The patient did not develop any further complications and was subsequently discharged. The patient was followed after 1 month, and he gave consent for coronary angiography, which was normal [Figure 4]. Sewer gas poisoning leading to transient and focal ST-segment elevation in the ECG mimicking anterior wall myocardial infarction was seen in our case. Such a localized effect on the myocardium has never been reported to the best of our knowledge.

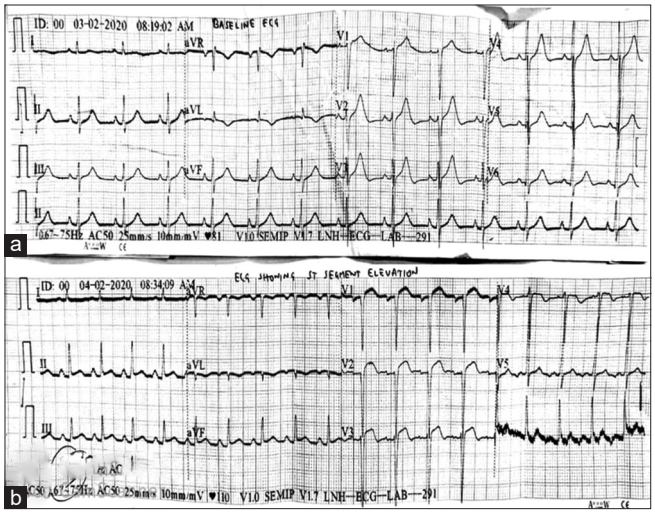

Figure 1: (a) Normal baseline ECG of the patient. (b) Day 3 ECG showing focal ST-segment elevation in the anteroseptal leads in V1 to V3

Discussion

Diagnosis of hydrogen sulphide poisoning lies primarily on history and detailed clinical examination as neuroimaging modalities are non-specific, showing cerebral oedema or ischemia involving the cerebral cortex and hippocampus. There is a role for analysis of sulphide or thiosulphate (a metabolite of sulphide) in the blood and urine of the patients. In our case, both the workers became unconscious immediately on entering the sewer, with one of them dying on the spot, suggesting that both were exposed to very high levels of this gas. Delayed neurological sequela has been reported, such as poor memory and neuropathies leading to weakness. Such issues were not encountered in the present case.

Sewer gas can lead to both left ventricular systolic dysfunction and chemical pneumonitis, both of which contribute to the pathophysiology of the development of ARDS.[6] In our case also, the patient developed reduced cardiac output along with ARDS. ARDS can be explained because of direct toxicity of various gases or because of direct effects on the heart leading to reduced contractility of the myocardium, further increasing alveolar oedema. In our case, the patient developed ST-segment elevation in anteroseptal leads V1 to V3, along with hypokinesia of the anterior wall. This transient ST-segment elevation was associated with increased CPK levels and Troponin T positivity with rapid diagnostic kits. Of particular note was the rapid recovery of the patient with ECG returning to the baseline along

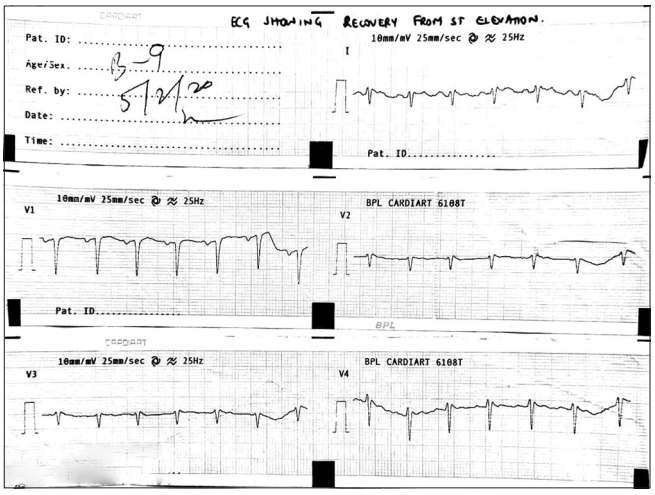

Figure 2: Day 5 ECG showing return to the baseline

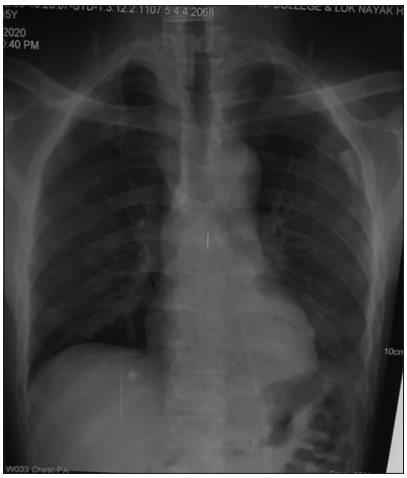

Figure 3: Chest x-ray of the patient showing mild interstitial opacities post recovery from ARDS

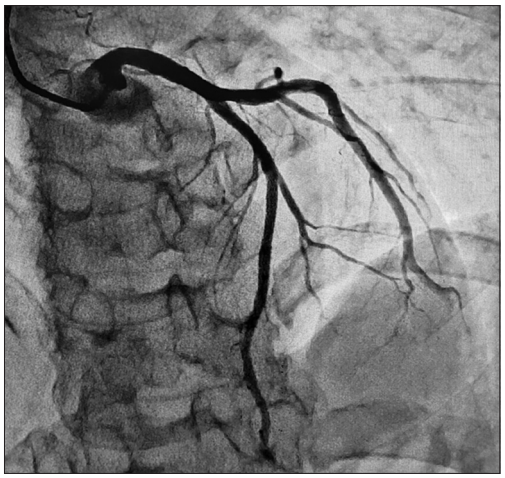

Figure 4: Coronary angiogram of the patient after 1 month showing normal epicardial coronaries

with normalisation of the left ventricular ejection fraction along with CPK enzyme levels.

The role of nitrite therapy, including 3% sodium nitrite or amyl nitrate, has been seen in a few cases. This must be given as early as possible.[7] The authors suggest that nitrite therapy must be given to such patients; however, one must refrain from using it if a patient has a mean arterial pressure <65 mm of Hg or shows signs of nitrite toxicity. Hyperbaric oxygen therapy which might displace hydrogen sulphide from cytochrome oxidase remains a logical option.[8,9] Hydroxycobalamine, which is given for cyanide poisoning, may form a stable complex with H_2S, and it has been shown to reduce sulphide and thiosulphate concentrations in a case report[7], but its routine use remains controversial. In our patient, we initially considered giving sodium nitrite 300 mg given by slow IV push, but it could not be given as he developed shock; hence, we shifted our management to a completely conservative approach.

Conclusion

- In any case of acute unconsciousness in manual scavengers, the possibility of hydrogen sulphide poisoning must be kept in mind. It can present with neurological toxicity and cardiac depression with ARDS.
- In cases of acute poisoning, there are a few case reports of diffuse ST-segment elevation in the ECG.[10,11] However, focal ST-segment elevation involving one territory of the heart has not been known previously to the best of our knowledge.
- The focus of management should be first on supportive care with a possible role of sodium nitrite therapy provided the clinical condition of the patient permits. Blood and urine sulphide and thiosulphate levels should be measured if available.
- There must be follow-up of these patients to study the delayed neurocognitive deficits which may develop later on in their illness.
- Because of the large number of personnel involved, a registry of such cases is required, which may include their data for analysis and research in the disciplines of critical care and toxicology for formulating appropriate management guidelines.

Limitations

We were unable to perform blood and urine sulphide and thiosulphate levels.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. MILBY TH. Hydrogen sulfide intoxication. Review of the literature and report of unusual accident resulting in two cases of nonfatal poisoning. J Occup Med 1962;4:431-7.
2. Vannatta JB. Hydrogen sulfide poisoning. Report of four cases and brief review of the literature. J Okla State Med Assoc 1982;75:29-32.
3. Haouzi P, Sonobe T, Judenherc-Haouzi A. Hydrogen sulfide intoxication induced brain injury and methylene blue. Neurobiol Dis 2020;133:104474.
4. Belley R, Bernard N, Côté M, Paquet F, Poitras J. Hyperbaric oxygen therapy in the management of two cases of hydrogen

sulfide toxicity from liquid manure. CJEM 2005;7:257-61.

5. Knight LD, Presnell SE. Death by sewer gas: Case report of a double fatality and review of the literature. Am J Forensic Med Pathol 2005;26:181-5.
6. Chaari A, Bahloul M, Chelly H, Sahnoun M, Bouaziz M. Défaillance cardiaque et neurologique secondaire à une intoxication accidentelle par l'hydrogène sulfuré: à propos d'un cas. Ann Fr Anesth Reanim 2010;29:304-7.
7. Fujino Y, Inoue Y, Onodera M, Kikuchi S, Endo J, Endo S, *et al.* Case followed by delayed loss of consciousness after exposure to hydrogen sulfide that was treated with intermittent administration of sodium nitrite. Chudoku Kenkyu 2010;23:297-302.
8. Smilkstein MJ, Bronstein AC, Pickett HM, Rumack BH. Hyperbaric oxygen therapy for severe hydrogen sulfide poisoning. J Emerg Med 1985;3:27-30.
9. Price WD, Price SM, Johnston M. Novel treatment of dihydrogen sulfide inhalation using hyperbaric oxygen during mass casualty. Undersea Hyperb Med 2021;48:153-6.
10. Yang Y, Hu D, Peng D. Diffuse ST-segment elevation after hydrogen sulfide intoxication. J Emerg Med 2018;54:241-3.
11. Amino M, Yoshioka K, Suzuki Y, Uemura S, Sakurai K, Fukushima T, *et al.* Improvement in a patient suffering from cardiac injury due to severe hydrogen sulfide poisoning: A long-term examination of the process of recovery of heart failure by performing nuclear medicine study. Intern Med 2009;48:1745-8.