

ARDS Induced by Mercury Vapors from Amalgam during Dental Procedure: A Case Report

AHMADREZA MASHREGHI¹, ANAHITA ALIZADEH GHAMSARI², ARMAN HAKEMI^{2,*}

¹M.D, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran

²Medical Toxicology Research Center, Mashhad University of Medical Sciences, Mashhad, Iran

Abstract

Background: Acute respiratory distress syndrome (ARDS) is a rare but severe complication of elemental mercury vapor inhalation. Dental amalgam, a source of mercury, poses an occupational and patient risk if not handled with appropriate precautions. We report a case of severe ARDS secondary to acute mercury vapor toxicity from a dental procedure.

Case Presentation: A 55-year-old male presented to the emergency department with acute respiratory distress that began shortly after a 10-minute exposure to dental amalgam vapors during a procedure. His initial urinary mercury level was markedly elevated at 853.2 µg/L. A chest CT scan confirmed severe ARDS, showing significant bilateral pulmonary involvement. The patient was admitted and immediately started on a treatment regimen including the chelating agent Succimer and broad-spectrum antibiotics to prevent secondary infection.

Discussion: After a seven-day inpatient course of chelation therapy, the patient showed significant clinical improvement. A follow-up chest CT revealed a marked reduction in the pulmonary lesions. He was discharged in stable condition with instructions to continue oral Succimer therapy for an additional two weeks to ensure complete systemic detoxification.

Conclusion: This case highlights that acute, high-intensity exposure to mercury vapor during dental procedures can lead to life-threatening ARDS. It underscores the critical importance of prompt diagnosis and immediate initiation of chelation therapy with agents like Succimer to mitigate pulmonary injury and prevent irreversible complications. Furthermore, this report emphasizes the necessity of stringent preventive safety measures in dental settings and the value of comprehensive follow-up to monitor for long-term sequelae of mercury toxicity.

Keywords: ARDS, Dental Amalgam, Mercury Vapors, Succimer, Chelation Therapy

How to cite this article: Mashreghi A, Alizadeh Ghamsari A, Hakemi A. ARDS Induced by Mercury Vapors from Amalgam during Dental Procedure: A Case Report. *Asia Pac J Med Toxicol* 2025; 14(3): 124-26.

INTRODUCTION

Mercury, a heavy metal of concern, exists in various forms and has a long history of use in diverse applications, including gold mining and the production of dental amalgams (1, 2). There are three distinct forms of mercury: elemental, organic, and inorganic. The elemental form is primarily absorbed through inhalation, after which it disperses extensively throughout the body and accumulates in multiple organs and tissues (3, 4). Inhalation of mercury vapor is considered the most toxic and detrimental form of mercury exposure, and it may result in pneumonitis, acute respiratory distress syndrome (ARDS), and potentially fatal outcomes (5). Although the use of dental amalgam in restorations is generally considered safe (6, 7), cases of

ARDS caused by inhalation of amalgam vapors during dental procedures are rare. This case report aims to elucidate the clinical manifestations and therapeutic interventions employed in managing a patient afflicted with severe ARDS subsequent to the inhalation of amalgam vapors and mercury toxicity during dental procedures.

CASE PRESENTATION

A 55-year-old male presented to the emergency department following known exposure to mercury vapor. The patient reported severe dyspnea occurring during a dental procedure, where he was exposed for approximately 10 minutes to vapor released from heated amalgam. Upon admission, the patient's vital signs were as follows: respiratory rate of 18 breaths per minute, pulse rate of 95

*Correspondence to: Dr. Aman Hakemi, MD, Fellowship of Clinical toxicology, Emergency Medicine specialist, Medical Toxicology Research Center, Mashhad University of Medical Sciences, Mashhad, Iran. Email: hakemia971@mums.ac.ir. Tel: +989153464197

beats per minute, and afebrile. His oxygen saturation (SpO₂) was 76%, prompting immediate administration of oxygen via a facial mask. A physical examination revealed bilateral coarse crackles on lung auscultation and the use of accessory respiratory muscles. No other clinical findings, including a neurological examination, were observed to be remarkable. The patient had no prior history of underlying pulmonary conditions. A lung CT scan revealed the presence of diffuse ground-glass opacities with reticulation and interlobular septal thickening, arranged in a patchy distribution (Figure 1). Laboratory tests revealed elevated urinary mercury levels of 853.2 µg/L and a white blood cell count of 16.09 × 10⁹/L, with 72.1% being neutrophils and 17.7% being lymphocytes. Conventional laboratory values, including those from arterial blood gas analyses, were within standard parameters.

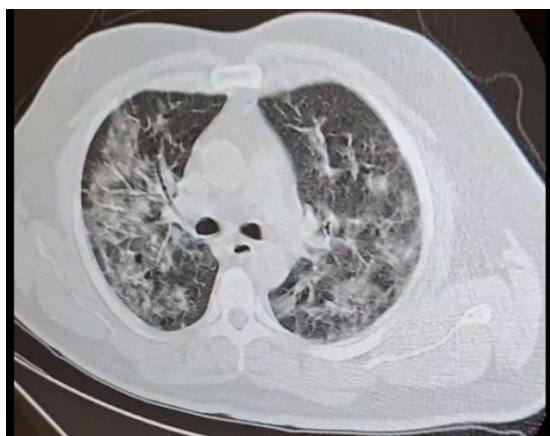


Figure 1. patient's HRCT scan indicating bilateral ground glass opacities with reticulation and interlobular septal thickening

Consequently, the patient was diagnosed with acute respiratory distress syndrome (ARDS) secondary to mercury exposure, potentially accompanied by pneumonia. The therapeutic approach involved the administration of intravenous meropenem and vancomycin, each at a dosage of 1 g every 12 hours, in conjunction with chelation therapy utilizing Succimer at a dose of 400 mg thrice daily. Following a 48-hour treatment period, during which the patient exhibited negative procalcitonin levels and no confirmed bacterial source, the antibiotic regimen was transitioned to oral levofloxacin at a dosage of 750 mg once daily. During the patient's subsequent hospital stay, there was a marked improvement in clinical status, with the resolution of dyspnea. On the seventh day of admission, the patient's oxygen saturation improved to 92%, and lung auscultation revealed only mild rhonchi. Subsequent follow-up HRCTs demonstrated a modest decrease in the extent of pulmonary lesions. Consequently, the patient was discharged with a prescription for oral levofloxacin, oral

Succimer, and a Salmeterol and Fluticasone inhaler. Three weeks after the initiation of chelation therapy, the patient remained asymptomatic, and urinary mercury levels had decreased to 12.5 µg/L.

DISCUSSION

This case report describes a patient with acute respiratory distress syndrome (ARDS) caused by mercury vapor inhalation during dental procedures. Dental amalgam, a compound used in dental restoration, is composed of approximately 50% elemental mercury (7), and as observed in this case, heating it accelerates the vaporization of mercury (8, 9). The health implications of mercury vapor are significant due to its colorless and odorless nature, allowing for easy inhalation and rapid absorption into the body (2, 7). The inhalation of high doses of elemental mercury can result in severe pulmonary manifestations, as approximately 80% of inhaled elemental mercury is absorbed through the lungs, leading to rapid cellular damage. The clinical progression of mercury vapor poisoning can be categorized into three distinct phases. The initial phase is characterized by the manifestation of flu-like symptoms. The intermediate phase is distinguished by the emergence of severe respiratory symptoms. The late phase resembles the clinical findings of acute respiratory distress syndrome (ARDS) and is further marked by persistent central nervous system (CNS) symptoms, while complaints related to other organ systems tend to resolve (10, 11). Given the approximately two-month half-life of elemental mercury (12), periodic follow-ups are essential in the patient described in this case to monitor for potential CNS involvement.

The management of acute mercury poisoning entails the cessation of exposure, the provision of supportive care, and the implementation of measures to facilitate the elimination of mercury from the body. In cases of pneumonitis caused by acute elemental mercury exposure, patients may require supplemental oxygen, close monitoring of their respiratory status, and mechanical ventilation to address respiratory failure effectively (13). Chelation therapy is advised for symptomatic patients with 24-hour urinary mercury levels of 100 µg/L or higher (14). Chelating agents, such as penicillamine, dimercaprol (British Anti-Lewisite), 2,3-dimercapto-1-propanesulfonic acid (DMPS), and meso-2,3-dimercaptosuccinic acid (DMSA), have been demonstrated to be effective in promoting mercury excretion through urine and reducing blood mercury concentrations (15). In this particular case, treatment with Succimer was administered over a 21-day course. A critical aspect of managing patients with mercury-induced toxicity pertains to the timing of chelation therapy. While there exists limited evidence to substantiate whether chelation therapy enhances early or late pulmonary outcomes in mercury-induced pneumonitis, prompt initiation of treatment is likely to augment its efficacy. Animal studies indicate that delays in chelation therapy significantly diminish its effectiveness, particularly concerning renal outcomes. Consequently,

treatment initiation should be prompt, ideally within minutes to a few hours after exposure, as efficacy diminishes with delay. A study by Rowens et al. reported four patients with ARDS due to mercury vapor exposure during amalgam handling, all of whom ultimately succumbed to the condition. A salient distinction in the management of this case is the timing of chelation therapy. In this case, treatment was initiated within hours due to a high index of suspicion for mercury poisoning. In contrast, in the Rowens study, therapy was delayed until six days after symptom onset. This observation underscores the importance of early chelation therapy in potentially preventing severe complications and improving patient outcomes.

In this case, a critical aspect that must be addressed is the prevention of exposure to toxic levels of mercury vapor for both the patient and the dental practitioner. Mercury begins to evaporate at 20°C, with its volatility significantly increasing as the temperature rises, reaching up to eight times higher levels at 50°C (16, 17). In environments subject to temperature fluctuations, the concentration of mercury in the air can fluctuate between 0.1% and 10%, depending on the extent of temperature increase (18, 19). Therefore, adherence to standardized guidelines for the safe handling and use of dental amalgam is imperative to minimize its toxic effects and safeguard the health of individuals involved in dental procedures.

The limitations of study were: The study used a single dose level and treatment duration, and a rats model that may not accurately represent human physiology.

CONCLUSION

This case underscores the critical health risks associated with mercury vapor inhalation, particularly during dental procedures involving the handling of amalgam under conditions of elevated temperature. Acute exposure can rapidly progress to severe pulmonary manifestations, including acute respiratory distress syndrome (ARDS). The prompt recognition of mercury toxicity and the early initiation of chelation therapy, as demonstrated in this case, are pivotal in mitigating complications and improving outcomes. To this end, adherence to safety protocols for dental amalgam use remains a cornerstone of preventing exposure to mercury for patients and practitioners alike.

Ethical consent: The patient provided written informed consent for the publication of this report, and the corresponding author was responsible for obtaining this consent.

Conflict of interest: The authors declare no conflict of interest.

Funding and Support: The authors declare that there are

no funding or financial sources relevant to this work, nor any conflicts of interest.

REFERENCES

1. Bernhoft RA. Mercury toxicity and treatment: a review of the literature. *J Environ Public Health*. 2012;2012(1):460508.
2. Kamensky OL, Horton D, Kingsley DP, Bridges CC. A case of accidental mercury intoxication. *J Emerg Med*. 2019;56(3):275-8.
3. Gul Oz S, Tozlu M, Yalcin SS, Sozen T, Sain Guven G. Mercury vapor inhalation and poisoning of a family. *Inhal Toxicol*. 2012;24(10):652-8.
4. Zhang H, Huang S, Wang D. A case of acute mercury vapour poisoning. *Postgrad Med J*. 1975;51:428-30.
5. Sarikaya S, Karcioğlu O, Ay D, Cetin A, Aktas C, Serinken M. Acute mercury poisoning: a case report. *BMC Emerg Med*. 2010;10:1-3.
6. Rathore M, Singh A, Pant VA. The dental amalgam toxicity fear: a myth or actuality. *Toxicol Int*. 2012;19(2):81.
7. Clarkson TW, Magos L. The toxicology of mercury and its chemical compounds. *Crit Rev Toxicol*. 2006;36(8):609-62.
8. Caravati EM, Erdman AR, Christianson G, Nelson LS, Woolf AD, Booze LL, et al. Elemental mercury exposure: an evidence-based consensus guideline for out-of-hospital management. *Clin Toxicol*. 2008;46(1):1-21.
9. Ozuah P. Mercury poisoning. *Curr Prob Pediat* 30(3): 91–99. 2000.
10. Divecha C, Agarwal S, Tullu MS, Deshmukh CT, Agrawal M, Shaikh SS. Acute respiratory distress syndrome caused by mercury inhalation: A case report. *J Pediatr Intensive Care*. 2015;4(03):168-70.
11. Ellenhorn M. Mercury poisoning In: Ellenhorn MJ, ed. *Medical toxicology*. 1997:1588-602.
12. Risher JF, Nickle RA, Amler SN. Elemental mercury poisoning in occupational and residential settings. *Int J Hyg Environ Health*. 2003;206(4-5):371-9.
13. Ozuah PO. Mercury poisoning. *Curr Probl Pediatr*. 2000;30(3):91-9.
14. Ye B-J, Kim B-G, Jeon M-J, Kim S-Y, Kim H-C, Jang T-W, et al. Evaluation of mercury exposure level, clinical diagnosis and treatment for mercury intoxication. *Ann Occup Environ Med*. 2016;28:1-8.
15. Hammerling J, Kanters A, Jacobs B, Franzblau A, Park PK, Napolitano LM. An Unusual Cause of Severe Hypoxemia and Acute Respiratory Distress Syndrome. *Chest*. 2020;158(2):e71-e7.
16. Hylander LD, Meili M. 500 years of mercury production: global annual inventory by region until 2000 and associated emissions. *Sci Total Environ*. 2003;304(1-3):13-27.
17. Dalla Costa R, Cossich ES, Tavares CRG. Influence of the temperature, volume and type of solution in the mercury vaporization of dental amalgam residue. *Sci Total Environ*. 2008;407(1):1-6.
18. Iverfeldt Å, Lindqvist O. Atmospheric oxidation of elemental mercury by ozone in the aqueous phase. *Atmos Environ* (1967). 1986;20(8):1567-73.
19. Chan KC, Svare CW. Mercury vapor emission from dental amalgam. *J Dent Res*. 1972;51(2):555-9.