

Homicidal Acute Formalin Poisoning in an Infant From a Rural Sericulture Family Presenting With Multisystem Failure

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Abstract: Acute poisoning of formalin is rare because of its strong irritating effect and alarming odor. Although few cases of acute poisoning in adults have been reported in literature, to our knowledge, this is the first case report of formalin poisoning in an infant presenting with multisystem failure. Despite proper supportive treatment in the absence of antidote, the infant died within 13 hours after deliberate poisoning.

Key Words: formalin, formaldehyde, poisoning, infant, multisystem failure

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Formalin is a 40% solution of formaldehyde (HCHO) in water. Formaldehyde is a colorless, water-soluble, irritating, pungent, highly soluble gas and is absorbed from all the surfaces of body.¹

Acute poisoning of formalin is rare owing to its strong irritating effect and alarming odor. Very few cases of acute poisoning in adults have been reported in literature. Little is known about the clinical effects of formalin poisoning because quite a few cases have reached hospital before death in acute poisoning. We report the clinical presentation, management, and prognosis of formalin poisoning in a 6-month-old infant, which, to our knowledge, has not been reported in the literature.

CASE

A 6-month-old girl was brought to the emergency department with complaints of altered sensorium and breathing difficulty. Her condition was apparently normal a couple of hours before. Her mother gave a history of deliberate poisoning by herself with approximately 10 to 15 mL of a disinfectant used for silk farming in rural India.

The patient was the second child born to a nonconsanguineous married parents, with rural background, weighing 3150 g at term by uncomplicated vaginal delivery in a district hospital. She was exclusively breast-fed and had received appropriate immunization with normal developmental milestones till date. The patient started to have convulsions during the initial clinical assessment.

On examination, her heart rate was 160 beats/min, respiratory rate was 72 breaths/min, oxygen saturation was 77% in air, and her Blantyre coma score was 2 to 3 of 5. Her pupils

were bilaterally symmetrical and reactive to light. There was generalized hypertonia of the body. She had intercostal and subcostal recessions and bilateral coarse crepitations throughout the lung fields. Heart sounds were normal with no murmurs, and peripheral pulses were feeble. The abdomen was soft with no clinical abnormalities. The possibility of formalin poisoning was suspected.

Gastric aspiration was done with the nasogastric (NG) tube, and the aspirate was blood mixed with fluid of about 5 to 6 mL. The stomach contents were diluted with water as a first aid measure. This was followed by administration of charcoal. The patient was placed in a warm couch and was supplied oxygen with nasal prongs. Adequate intravenous fluids were administered along with sodium bicarbonate and vasopressors.

Later, the poison was confirmed to be formalin as the mother brought the container that was clearly labeled by the manufacturing company. Chemical analysis of the gastric aspirate of the patient and the fluid in the container brought by the mother further confirmed formalin poisoning.

Routine hematological investigations revealed no significant abnormality. Arterial blood gas analysis showed metabolic acidosis with a high anion gap ($\text{PaO}_2/\text{FiO}_2$, 82). Liver function test results were as follows: alanine aminotransferase, 102 U/L (reference range, 15–55 U/L); aspartate aminotransferase, 119 U/L (reference range, 5–45 U/L); total bilirubin, 0.8 mg/dL (reference range, 0.2–1.5 mg/dL); and direct bilirubin, 0.6 mg/dL (reference range, 0.5–0.6 mg/dL). Renal function test results were as follows: serum creatinine, 0.97 mg/dL (reference range, 0.2–0.4 mg/dL); blood urea, 17 mg/dL (reference range, 5–18 mg/dL); serum potassium, 5.6 mEq/L (reference range, 3.5–5.0 mEq/L); serum sodium, 142 mEq/L (reference range, 135–145 mEq/L); and serum calcium, 9.5 mg/dL (reference range, 8.8–10.2 mg/dL). The level of blood formic acid was significantly increased (38 mg/L). A chest radiograph (Fig. 1) revealed multiple bilateral fluffy infiltrates in the lung fields, with normal cardiac silhouette. The final diagnosis of multisystem failure (respiratory, cardiovascular, nervous, and renal systems) due to formalin poisoning was made.

On review 1 hour later, the patient's level of consciousness had decreased (Blantyre coma score, 1–2/5) and oxygen saturation was decreased to 72% in air, with increased respiratory distress. Bilateral coarse crackles were increased. Her pupils were normal and reactive; there was mild neck stiffness and her limbs were hypertonic bilaterally. Urine output was very minimal, with raised serum creatinine level. The patient was intubated and put on ventilator with all the supportive measures. Despite hemodialysis and proper treatment measures, her condition deteriorated rapidly and died during the 11 hours of hospital stay.

DISCUSSION

Formalin ingestion is rare because of its strong odor and highly irritant even at its low concentration; however, there are only few documented cases of accidental, homicidal, and

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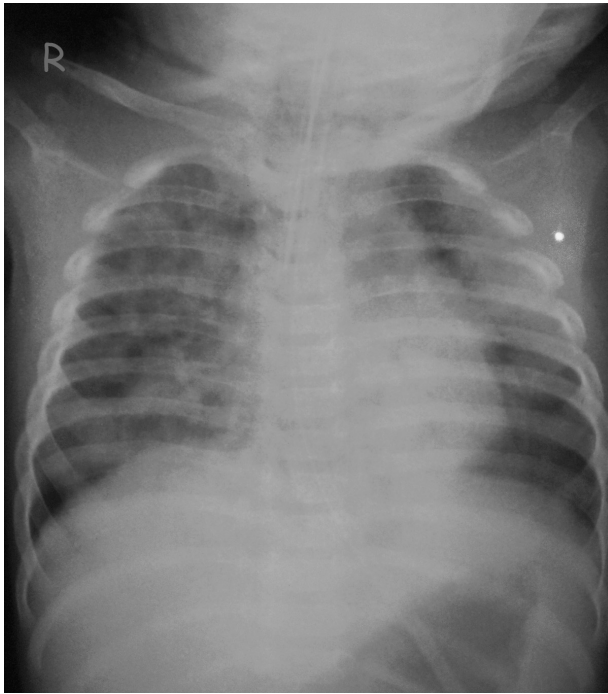


FIGURE 1. Chest radiograph (frontal view) of the 6-month-old infant showing multiple bilateral fluffy infiltrates in the lung fields, with normal cardiac silhouette consistent with acute respiratory distress syndrome.

suicidal acute poisoning have been reported. To our knowledge, this is the first case report of formalin poisoning in an infant presenting with multisystem failure.

Formalin is used for various purposes in domestic, industrial, and medical fields. Common uses of the formalin in medicine are preservation of surgical specimens, treatment of uncontrolled intravesical hemorrhage, and prevention of hydatid cyst dissemination and radiation-induced hemorrhagic proctitis.² The common domestic use of formalin in rural areas is as a disinfectant to fumigate the silkworm growing room in sericulture.

Formaldehyde is rapidly converted to formic acid in the body with the help of formaldehyde dehydrogenase present in the liver and erythrocytes. Metabolic acidosis rapidly develops because of the relatively slow metabolism of the formic acid into carbon dioxide and water.³ Absolute toxic levels of the formic acid are not known; however, the normal levels of blood and urine range from 0 to 12 mg/L and 0 to 27 mg/L, respectively.⁴ The fatal adult dose of formalin is about 60 to 90 mL and far less is needed in the pediatric age group like our patient.⁵ Gastrointestinal symptoms of formalin ingestion include nausea, burning in the mouth and esophagus, hemoptysis, vomiting of tissues, abdominal pain, and diarrhea. Formic acid is highly toxic to the cells of the liver, kidney, and brain, which can cause jaundice, albuminuria, hematuria, anuria (due to acute tubular necrosis), metabolic acidosis, convulsions, central nervous system depression, unconsciousness, and death due to cardiovascular failure.⁶

Formalin can cause respiratory symptoms of variable severity including pneumonitis and acute respiratory distress syndrome. It may cause aspiration pneumonitis due to increases secretions due to the irritant action of the formalin.⁷

Interestingly, children are more sensitive to formaldehyde toxicity than adults and are associated with greater impact on the lung function in inhalational toxicity.⁸

Treatment of formalin poisoning is supportive because there is no antidote. First and foremost is that emesis should not be induced. Gastric contents are diluted with water or milk followed by charcoal (1 g/kg) or 1% ammonium carbonate.^{2,9} Milk is preferred over water because it dilutes and neutralizes the poison whereas water only dilutes. The effective binding of charcoal with formaldehyde is unknown, but it is helpful if administered within 1 hour after ingestion following lavage (dose of 25–50 g in children). Preferably, charcoal administration is avoided because it could obscure endoscopic findings.

Gastric lavage can be considered if a patient has ingested a large dose and presents within 30 minutes after ingestion. Nasogastric tube may further injure the chemically damaged esophagus or stomach and should be inserted with extreme care. Endoscopy may be done to evaluate the extent of corrosive injury to upper gastrointestinal tract. Lavage is discouraged in children because of the risk of perforation from the NG tube. Lavage is performed after intubation under endoscopic guidance in children. Increased throat swelling may require endotracheal intubation and cricothyroidotomy.¹⁰

Patient should be kept warm, and vitals are monitored continuously. Close monitoring of the serial blood counts, electrolytes, acid-base disturbances, and renal and liver function tests is recommended. Signs of gastrointestinal hemorrhage should be watched out.

Adequate fluids are administered for the hypotension along with sodium bicarbonate and vasopressors. Sodium bicarbonate acts as vasopressor, decreases formation of formate, and prevents penetration of the undissociated form of formic acid into the cerebrospinal fluid.² Hemodialysis is indicated in case of deteriorating vital signs, reduced urine output, and severe metabolic acidosis. Hemodialysis can also be considered in patients having good urinary output with inadequate response to alkali treatment—it manages acute tubular necrosis and metabolic acidosis by effectively removing the methanol, formic acid, and formate.^{10,11} Airway and ventilation support must be done if the patient has lost consciousness and if there is reduced oxygen saturation.

CONCLUSIONS

Formalin is a substance commonly used for various purposes and causes potential life-threatening complications even if only a small quantity was ingested. Fatal dose may be less in children and may cause multisystem failure and death within a short span of time. Adequate supportive measures and a multidisciplinary approach are required for the effective management in the absence of an antidote. In general awareness of the toxicity, proper storage and labeling of formalin may prevent the unwanted hazard.

REFERENCES

1. Koppel C, Baudisch H, Schneider V, et al. Suicidal ingestion of formalin with fatal complications. *Intensive Care Med.* 1990;16:212–214.
2. Pandey CK, Agarwal A, Baronia A, et al. Toxicity of ingested formalin and its management. *Hum Exp Toxicol.* 2000;19:360–366.
3. Boeniger MF. Formate in urine as a biological indicator of formaldehyde exposure: a review. *Am Ind Hyg Assoc J.* 1987;48:900–908.

4. Myers JA, Mall J, Doolas A, et al. Absorption kinetics of rectal formalin instillation. *World J Surg.* 1997;21:886–889.
5. Sidhu KS, Sidhu JS. An alleged poisoning with methanol and formaldehyde. *Vet Hum Toxicol.* 1999;41:237–242.
6. Hawley CK, Harsch HH. Gastric outlet obstruction, as a late complication of formaldehyde ingestion: a case report. *Am J Gastroenterol.* 1999;94:2289–2291.
7. Patterson WP, Craven DE, Schwartz DA, et al. Occupational hazards to hospital personnel. *Ann Intern Med.* 1985;102:658–680.
8. Krzyzanowski M, Quackenboss JJ, Lebowitz MD. Chronic respiratory effects of indoor formaldehyde exposure. *Environ Res.* 1990;52:117–125.
9. Lund A. Rational treatment of formaldehyde poisoning. *Acta Pharmacol Toxicol (Copenh).* 1947;3:323–329.
10. U.S. Department of Health and Human Services. Formaldehyde: toxic substances portal. Agency for Toxic Substances and Disease Registry (ATSDR) Web site. Available at: <http://www.atsdr.cdc.gov/tfacts111.pdf>. Accessed March 15, 2012.
11. Fair WR. Formalin in the treatment of massive bladder hemorrhage. Techniques, results and complications. *Urology.* 1974;3:573–576.