Accidental formalin poisoning in a child with acute fatal manifestations: A rare case report

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ABSTRACT

Formalin is a protoplasmic poison, which poses a potential occupational hazard among morticians, embalmers, pathologists, and hospital staff. The crystal-clear appearance of formalin can be easily mistaken for normal saline, local anesthetics, hydrogen peroxide, sodium hypochlorite, and spirit in health care facilities and water in domestic settings. However, accidental poisoning is extremely rare because of its low olfactory threshold, strong irritant nature, pungent taste, and odor. This is also evident from the scarce scientific literature on this topic. Here, we presented a case of accidental, fatal formalin ingestion by a 4-year-old child who succumbed to the poisoning within 12 h of ingestion. The case presented here is unique because of its rarity in causing accidental poisoning by ingestion and first of its kind in a child.

Keywords: Accidental poisoning, formaldehyde, formalin, formalin ingestion, pediatric poisoning

Introduction

Accidental poisoning is one of the most common preventable causes of morbidity and mortality among the pediatric age group. Approximately 80% of pediatric poisoning occurs in children of 1 to 5 years of age due to rapid neurocognitive development, curiosity, hyperactivity, need to explore more, the tendency to mouthing objects, and difficulty differentiating poisonous from nonpoisonous substances.¹,² Household chemicals such as toilet cleaning agents, kerosene, and pesticides are the most commonly reported substances involved in these poisonings.¹,³

Formalin is an aqueous formaldehyde solution; the concentration may typically vary from 30% to 50%, along with 15% methanol added as a stabilization agent to prevent polymerization.³⁴ It is widely used in medical, dental, and veterinary sciences as a tissue fixative for histopathological examination (10% neutral buffered solution), disinfecting agent, and constituent of embalming fluid.³⁴ Besides, formalin is also used as a parasiticide, fungicide, algicide in fish industries⁹ and as a fumigant in poultry farms for disinfection.¹⁰ Formalin is an unusual poison among children and adults because of its crystal-clear or colorless nature, it can be easily mistaken for normal saline, local anesthetics, hydrogen peroxide, sodium hypochlorite, a spirit in health care facilities, and water in domestic settings.

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Only a few studies in the literature deal with accidental formalin poisoning in the context of accidental mishaps in dentistry and eye surgery following formalin injection instead of local anesthetic agents (lignocaine). Nevertheless, there was no accidental ingestion of formalin reported among the pediatric age group after a careful review of the literature. Knowledge of formalin poisoning is imperative for primary care providers as they play a crucial role in the management of this uncommon, yet fatal poisoning. Here, we present the first case of fatal, acute pediatric poisoning with accidental ingestion of formalin to the best of our knowledge.

**Case History**

A 4-year-old female child was referred to the emergency department with an alleged history of ingestion of an unknown quantity of formalin stored in a transparent water bottle in her father’s poultry farm. After ingestion, she had a single episode of vomiting before reaching a primary hospital. Gastric lavage was done at the primary hospital, then referred to our tertiary care center. Four hours after ingestion, she was presented with drowsiness, irritability, respiratory distress, cold extremities. Her vitals were as follows: Glasgow Coma Scale (GCS) score: 8/15 (Eye response-2, Verbal response-2, Motor-4), blood pressure: 84/50 mmHg, respiratory rate: 34 per min, the pulse rate: 178 beats per min, and SpO₂: 99% at room air. Bilateral pupils were mid-dilated and reactive to light.

Her comprehensive laboratory workup revealed the following: pH = 7.183, pCO₂ (kPa, mmHg): 16.6, Bicarbonate (HCO₃⁻): 6.3 mmol/L, Sodium: 139.2 mmol/L, Potassium: 4.9 mmol/L, Chloride: 109.2 mmol/L, Lactate: 4.5 mmol/L, and anion gap: 23.7 mEq/L suggestive of high anion gap metabolic acidosis. The blood glucose level was 158 mg/dL, and the measured urine output was only about 50 mL.

Supportive treatment consisting of intravenous fluids, sodium bicarbonate, antibiotics, antacids, antiemetics, and vasopressors were administered. However, her condition deteriorated after 6 hours of admission for which intubation was performed. Despite all efforts, she succumbed to the poisoning after about 12 h of ingestion. Clinically, refractory shock with severe metabolic acidosis due to formalin poisoning was certified as a cause of death. The body of the deceased was sent to the mortuary for postmortem examination.

An autopsy was performed approximately after 12 h of death. External examination revealed a moderately built and nourished girl with a height of 90 cm and 11.6 kg. The face was congested, and nail beds were cyanosed. Lips were bluish, dry, and cracking in appearance. Rigor Mortis was present throughout the body. Purplish postmortem lividity was present over the back of the body except over pressure areas and was fixed. There were no external injuries present on the body.

Exploration of the gastrointestinal tract revealed the thickened, leathery, congested, and edematous mucosa of the pharynx, epiglottis, and esophagus without ulcer and hemorrhage. However, mucosa of the stomach and parts of the small intestine was dark reddish-brown with diffuse ulcer and hemorrhage [Figure 1]. The stomach wall was thickened, firm in consistency (leathery), and edematous with translucent serosa. The stomach contained about 200 mL of brownish liquid that emitted a faint formalin odor and curdy-white material at places. There was no evidence of loss of rugosities and no perforation of the stomach wall [Figure 1]. However, peritoneal cavity examination showed about 200 mL of straw-colored fluid. Furthermore, about 100 mL of straw-colored fluid was found in each pleural cavities on thoracic cavity examination. Both lungs were congested and edematous. Multiple superficial epicardial hemorrhages were present over the posterior wall of the left ventricle. Both kidneys and liver showed severe congestion and edema on the cut section. The rest of the internal examination was unremarkable except for congestion of visceral organs. Parts of the stomach wall, heart, pieces of both lungs, liver, and kidneys were preserved for histopathological examination.

The histopathological examination of the stomach revealed sloughing of gastric mucosa, submucosal edema, hemorrhage, and necrosis of mucosal and submucosal layers with infiltration of acute inflammatory cells [Figure 2]. The liver showed large areas of periportal and bridging necrosis. Lung showed features of congestion with acute inflammatory infiltration. Given the history, clinical, autopsy, and histopathological findings, the cause of death was ascertained to be multiorgan failure resulting from severe metabolic acidosis of formalin poisoning.

**Discussion**

Formalin acts as a strong irritant or corrosive agent. Because of neutral pH, formalin is not classified under either acids or alkali. It acts as a fixative by precipitating proteins. Mechanism of action resembles methanol, as formalin is metabolized into formic acid.
As there is a high index of suspicion for unknown poisoning cases as they are the first point of care, primary care providers play a vital role in the management of formalin poisoning. A fatal dose of formalin ranges from 50 to 100 mL. A fatal dose of 20–50 mL with a fatal period of 7 h was also reported in the literature.[14] In one successfully managed case reported by Vos et al., ingestion of about 20 mL also resulted in multi-organ failure and shock. In our case, the exact quantity of ingestion could not be ascertained. However, from the detailed history and clinical findings, the quantity might probably be two mouthfuls of a child, approximately 20–30 mL.[18]

Patients with acute ingestion may present with burns over the mouth, pharynx, epiglottis, esophagus, and stomach, which may mimic acute abdomen. Systemic effects such as shock, acute respiratory failure, seizures, or coma are also observed if the presentation is delayed.[8,12,19] Presence of prominent tachycardia, severe metabolic acidosis, acute respiratory distress syndrome, acute renal failure, and anuria demonstrates severe toxicity in the present case.[8,12,19] Immediate decontamination of the exposed parts of the body with continuous irrigation of water is recommended. Emesis should not be induced, whereas about 120 to 240 mL water or milk will be preferable. Gastric lavage with a nasogastric tube should be done. The role of activated charcoal is inconclusive. As there is no antidote available for formalin poisoning, supportive therapy with IV fluids, bronchodilators, vasopressors, and correction of metabolic acidosis with bicarbonates are the mainstay of management.[10]

In addition, hemodialysis should be preferred to remove formic acid from the blood and correct metabolic acidosis, preventing circulatory collapse and acute tubular necrosis.[4,13,19] Invasive mechanical ventilation is also recommended, given impending respiratory failure from severe metabolic acidosis.[19] Infusion of N-acetyl cystine (NAC) is advocated to reduce formaldehyde conversion to formic acid, thereby preventing severe metabolic acidosis.[8] In addition, folic acid will be helpful to accelerate the conversion of formic acid into carbon dioxide and water.[19] On the other hand, methanol as a stabilizer in formalin contributes to the substantial morbidity and mortality in formalin poisoning. The high anion gap metabolic acidosis with elevated serum lactic acid level in the present case demonstrates the simultaneous presence of methanol poisoning. Ethanol (10%, IV) could be the drug of choice in treating methanol poisoning along with intravenous fluids and bicarbonates.[4]

The shock on arrival is regarded as the worst prognostic factor.[1] This was evident from the present case. Death may usually result from multi-organ failure, circulatory collapse, severe metabolic acidosis, acute renal failure, myocardial depression, global cardiac insufficiency, acute respiratory distress syndrome (ARDS) culminating in respiratory insufficiency.[8,12]

At autopsy, ingestion of formalin may be indicated by ulcer or erosion of the mucosa of the mouth, pharynx, esophagus, and stomach (antrum) shows sloughing of gastric mucosa, submucosal oedema, haemorrhage and necrosis of mucosal and submucosal layers with infiltration of acute inflammatory cells. Aldehyde dehydrogenase from the liver and red blood cells oxidizes formaldehyde to formic acid, which is further converted to carbon dioxide and water by a folate-dependent enzymatic reaction. In humans, relatively slow conversion of formic acid to carbon dioxide and water is suggested to precipitate accumulation of formic acid, which in turn leads to severe metabolic acidosis and systemic toxicity of formalin poisoning.[5,18]

Formic acid inhibits mitochondrial cytochrome oxidase and results in rapid coagulative necrosis of the systemic organs such as the heart, brain, liver, and kidneys immediately after being absorbed into the bloodstream.[18,19] It also exerts direct oxidant action on red blood cells (RBC), resulting in hemolysis.[14] The usual fatal dose of formalin ranges from 50 to 100 mL.[14,18] A fatal dose of 20–50 mL with a fatal period of 7 h was also reported in the literature.[16] In one successfully managed case reported by Vos et al., ingestion of about 20 mL also resulted in multi-organ failure and shock. In our case, the exact quantity of ingestion could not be ascertained. However, from the detailed history and clinical findings, the quantity might probably be two mouthfuls of a child, approximately 20–30 mL.[18]

Primary care providers play a vital role in the management of unknown poisoning cases as they are the first point of care in the majority of clinical settings. A high index of suspicion is required to make a timely diagnosis in formalin poisoning which is rarely encountered as accidental poisoning in children as well as adults. Clinically, formalin poisoning is suspected when a patient presented with burning pain involving the upper gastrointestinal tract with severe metabolic acidosis. Immediate arterial blood gas analysis is imperative to evaluate the anion gap and metabolic acidosis. The detailed history, circumstantial evidence, and associated clinical findings are critical in excluding other causes of metabolic acidosis such as renal failure, ethylene glycol or propylene glycol poisoning, aspirin or paracetamol poisoning, diabetic or alcoholic ketoacidosis.[21] The half-life of formaldehyde is only 90s, hence determination of formaldehyde concentration may be often unsuccessful.[19] Serum formic acid levels and methanol levels can be analyzed by gas chromatography/mass spectrometry (GC/MS), gas chromatography/flame ionization detector (GC/FID), and headspace gas chromatography (HSGC).[22] However, serum formic acid and methanol levels were not determined in the present case due to a lack of facilities.

Immediate decontamination of the exposed parts of the body with continuous irrigation of water is recommended. Emesis should not be induced, whereas about 120 to 240 mL water or milk will be preferable. Gastric lavage with a nasogastric tube should be done. The role of activated charcoal is inconclusive. As there is no antidote available for formalin poisoning, supportive therapy with IV fluids, bronchodilators, vasopressors, and correction of metabolic acidosis with bicarbonates are the mainstay of management.[10]

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stomach, and duodenum. As described above, the stomach might suffer the worst among other organs due to a longer duration of contact with formalin,[10] whereas the esophagus is affected minimally because of rapid passage.[12] Although the fixative property of formalin results in thickening or leathery stomach wall, perforation is not uncommon.[12] Chemical peritonitis without perforation is also suggested to occur due to stomach wall inflammation.[5] Loss of rugosities reported in the literature[5] was absent in our case. Pneumonia, hepatosplenomegaly, and jaundice were noted by Köppel et al.[12] were not seen in our case, likely due to the shorter duration of survival.

The previously reported cases in the literature were mainly from the dentistry following intraoral injection of formalin instead of local anesthetic agent (lignocaine)[7,14] or normal saline[15] for dental procedures. An isolated case of subcutaneous injection of formalin into all four eyelids before blepharoplasty was also reported.[8] Here, the reported child ingested formalin because of its colorless nature, resembling water filled in a transparent bottle, and preexisting pungent smell in the poultry environment. Inability to calculate the exact amount of ingested formalin and failure to analyze serum formic acid and methanol levels due to the lack of facilities are limitations in the present case.

**Conclusion**

The present case is unique because of the extreme rarity of formalin to cause the death of a child following accidental ingestion. The findings enlisted in this report reinforce the knowledge on formalin poisoning. Prompt diagnosis with aggressive supportive treatment remains the mainstay of management to prevent complications. Primary care physicians and pediatricians should be vigilant and suspect formalin poisoning in cases with a history of ingestion of colorless liquid, especially from a poultry setting. This report also highlights the need to regularize sales and storage of formalin in poultry farms. Furthermore, it is also stressed here that formalin should be stored in places inaccessible to the children using a child-proof special container with proper labeling of warning signs.

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**Conflicts of interest**

There are no conflicts of interest.

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