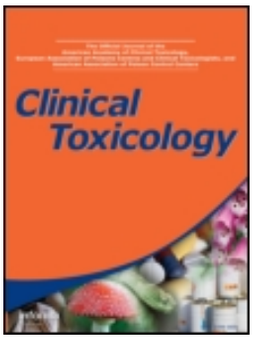

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CASE REPORT

Laryngeal edema and metabolic acidosis after Omnicide® ingestion

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Introduction. Glutaraldehyde and quaternary ammonium compounds are widely used as disinfectants and sterilizing agents. Glutaraldehyde is irritating to the eyes and upper respiratory tract, and has been associated with occupational asthma. Acute oral ingestion of a concentrated solution has not been previously reported in humans. *Case Report.* A 19-year-old woman presented after deliberate ingestion of a biocide containing glutaraldehyde and a quaternary ammonium compound. She developed respiratory distress and severe metabolic acidosis 10 hours after admission. Marked laryngeal edema was noted when she was being intubated. She eventually improved following supportive care and was discharged alive after 9 hospital days. *Discussion*. There are no reports of acute ingestions of both glutaraldehyde and quaternary ammonium compounds. As both these substances are known to cause metabolic acidosis, localized edema, erosion and sensitization of both the respiratory and alimentary tract. The clinical effect may be additive or synergistic. *Conclusions.* Omnicide ingestion should be closely monitored for metabolic acidosis and laryngeal edema which may progress to upper airway obstruction requiring urgent airway stabilization.

Keywords Glutaraldehyde; Metabolic acidosis; Laryngeal oedema

Introduction

Ominicide® is a poultry biocide containing a complex formulation of glutaraldehyde 15% w/v and coco benzyl dimethyl ammonium chloride 10% w/v. Glutaraldehyde (GA) is a low-molecular weight aliphatic dialdehyde used in industrial, scientific and biochemical applications. It is also used as an embalming fluid, a chemical intermediate, a fixative for tissues, for cross-linking protein and polyhydroxy materials, and tanning of soft leathers[1]. It is also used as a disinfectant and in sterilization of endoscopic and surgical instruments[2]. There are no published reports of oral GA poisoning. We describe a case of glutaraldehyde and quaternary ammonium compounds (Omnicide®) ingestion associated with laryngeal edema and severe metabolic acidosis.

Case report

A 19-year-old, previously healthy woman presented 1.5 hrs after the deliberate ingestion of 75ml of Omnicide®. Spontaneous vomiting occurred shortly afterwards. Her initial complaints were pain on swallowing and mild abdominal tenderness. No gastrointestinal decontamination procedures were performed. She looked well and was able to speak normally on admission. Her vital signs were pulse 88 beats/minute, supine blood pressure 130/90 mmHg, and respiratory rate of 23 breaths/minute. The remaining physical examination was unremarkable. Initial laboratory findings revealed a hemoglobin level of 10 g/dl, hematocrit 37%, white blood cell count of 11.3×10^9/L, serum sodium 140mmol/L, potassium 4.6mmol/L and normal hepatic transaminases. Ten hours after admission she developed dyspnea accompanied by a pulse of 126 beats/minute. Her blood pressure was 110/80mmHg, which remained stable throughout her illness. She had a respiratory rate of 31 breaths/minute, audible stridor, and a few bilateral basal crepitations. She was intubated and ventilated. During the intubation procedure laryngeal edema was noted. Arterial blood gas analysis after intubation revealed a pH of 7.11, pCO_2 of 12.4 mmHg, pO_2 of 162 mmHg, bicarbonate of 3.9 mEq/L, and a base excess of –25.6 mmol/L. An ECG showed sinus tachycardia and a chest x-ray was normal.

Intravenous antibiotics (ampicillin, metronidazole) and hydrocortisone therapy were started. Intravenous fluids with normal saline alternating with half-normal saline at a rate of 112mL/hour was given for the first 24 hours.

The patient's metabolic acidosis resolved within the first 24 hours with supportive care only. She did not receive any

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sodium bicarbonate. She had an uncomplicated course in the Intensive Care Unit and was extubated on the 6th day after ingestion. She did not undergo endoscopy because of her rapid clinical improvement. All medications were stopped and she was discharged on the 9th day after ingestion. She had continuing mild throat discomfort at discharge. A follow up visualization of the larynx was planned but she was lost to follow up.

Discussion

Reports of human exposure to GA and QACs are limited to a few individual cases [3–5] and there are no reports of acute ingestion. The lack of reports is likely due to its lack of common use in domestic or agricultural settings. Even weak solutions (2–3.5% in water) of GA are strong irritants to the skin or eyes. Prolonged exposure can produce localized edema and other symptoms suggestive of an allergic response [6]. Although its low vapor pressure reduces the exposure of GA via inhalation, GA vapor may act as an irritant to bronchial and laryngeal mucous membranes. Sensory irritant effects and sensitization of respiratory mucous membranes following chronic inhalation due to GA vapors has been reported [7–10].

Although exposure and consequential human health effects of skin, eye, and respiratory tract exposure to GA have been well documented [2,11–13], data on its acute oral toxicity is limited to animal studies. In acute oral toxicity testing in the rat, solutions less than 5% GA were of slight toxicity (LD_{50} range 3.34 to 12.30 ml/kg for 1 and 2% solutions)[13]. Concentrated solutions caused severe irritation and corrosion of the alimentary tract. Survivors recovered in 1 to 5 days of dosing and generally showed no gross pathology. Necropsy demonstrated gastric distention, congestion, hemorrhagic areas, thickening of the pyloric area, congested small intestine with variable congestion of the adrenal glands, liver, spleen, and lungs and scattered pulmonary hemorrhages [13]. None of these studies recorded metabolic acidosis as a complication. The paucity of oral animal toxicology studies reflects the fact that swallowing was regarded an unlikely route of human exposure[13].

Beauchamp et al. [1] suggested that GA is oxidized to semialdehyde and then to glutaric acid. Acidemia is hypothesized to result from glutaric acid accumulation. Little is known about the toxicologic effects of glutaric acid. Elevated glutarate levels in the urine or blood have been described in patients with certain metabolic disorders (e.g., glutaric acidosis and glutaric aciduria) and after ifosfamide overdose [14–18]. In glutaric acidosis, an autosomal recessive disorder characterized by macrocephaly, dyskinesia, dystonia, opisthotonos, choreoathetoid movements, and delayed development, severe metabolic acidosis results from glutaric acid accumulation due deficiency of glutaryl-CoA dehydrogenase[19,20]. After ifosfamide overdose, excessive urinary excretion of glutaric acid and sarcosine may occur, leading to defective mitochondrial fatty acid oxidation and acidemia [18]. Unfortunately, we were unable to obtain qualitative or quantitative confirmation of glutaraldehyde or its metabolites in this case, as laboratory facilities to measure these chemicals were not available.

Based on human exposures to formaldehyde, the laryngeal edema experienced by our patient after ingestion of GA may have been due to direct corrosive injury or subsequent inflammatory changes [21]; these have been observed in the upper airways after acute low-level exposure to formaldehyde. Moreover, delayed hypersensitivity leading to asthma has occurred 6 to 12 hours after occupational exposure to formaldehyde [22].

The systemic toxic mechanisms of quarternary ammonium compounds remain uncertain, but both antcholinesterase [23] and curare-like effects have been postulated [24]. Significant mucosal irritation and corrosion can occur depending on the volume of liquid ingested and the concentration of the compound. Concentrations of <5% may be mildly irritating, 5% to 10% moderately to severely irritating, and solutions >10% have caused ulceration, dermal necrosis [25] and metabolic acidosis [26].

The clinical presentation of throat pain, laryngeal edema, and metabolic acidosis is consistent with the patient's history of ingestion and the known properties of glutaraldehyde and a quaternary ammonium compound.

Metabolic acidosis in our case responded to adequate fluid therapy and supportive care, without neurological or nephrotoxic sequelae.

Conclusion

This case illustrates the local irritant and metabolic effects of Omnicide®. Oropharngeal injury can cause laryngeal edema, which could be life-threatening. Patients should be monitored closely for several hours after ingestion, as airway management with endotracheal intubation may be necessary.

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