Severe Esophageal Burn Following Chloral Hydrate Overdose in an Infant

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Chloral hydrate is generally considered to be a safe hypnotic drug, and is commonly used for short-term sedation before diagnostic procedures. Its irritant actions to the mucous membranes are usually limited. We report a rare complication of chloral hydrate overdose in an infant. An 8-month-old male infant became unconscious and required ventilation support after an overdose of chloral hydrate was administered to provide sedation for an ophthalmologic examination. White plaques and sloughing of the oropharyngeal mucosa were observed on the next day. Esophagogastroscopy revealed severe corrosive lesions on the whole esophagus. The child recovered after supportive treatment and his oral intake remained well without dysphagia after 1 year. This report illustrates the potential corrosive effect of chloral hydrate. Strict attention should be paid to the dosing and administration protocol of chloral hydrate in infants. The condition of the oropharyngeal mucosa should be carefully monitored after chloral hydrate administration. [J Formos Med Assoc 2006;105(3): 235–237]

Key Words: chloral hydrate, esophagus, overdose

Chloral hydrate is the oldest of the hypnotic depressants and is widely used to sedate children before diagnostic procedures. Although generally considered to be safe, adverse effects, such as central nervous system depression, respiratory depression, and cardiac arrhythmia, are well known.¹ Severe esophageal burn, however, has rarely been reported as an adverse effect of chloral hydrate ingestion. We report the case of an 8-month-old male infant who developed severe esophageal burn following chloral hydrate overdose.

Case Report

An 8-month-old male infant fell against the edge of the bed, causing his right eyelid to become swollen. He was brought to the ophthalmology clinic and chloral hydrate (50 mg/kg) was prescribed to sedate him for ophthalmologic examination. His body weight was 8.4 kg, but a dose of 8 g chloral hydrate instead of the correct dose of 0.4 g was inadvertently given orally. In addition, the chloral hydrate was insufficiently diluted with only 15 mL water. Ten minutes after ingesting the chloral hydrate, the patient lost consciousness and was transferred to the emergency room, where tracheal intubation and assisted mechanical ventilation were instituted. The patient was hypothermic (body temperature 35.4°C), his heart rate was 91 bpm, respiratory rate was 20/min, blood pressure was 57/11 mmHg, and oxygen saturation was 80%.

On physical examination, his extremities were flaccid, with decreased tendon reflex. Muscle tone was poor in all of the extremities. Glasgow Coma Scale score was E1M1VT. Pupils were constricted and unresponsive to light. No focal neurologic deficits were present. Widespread rales were
heard over both lungs and aspiration of vomited substance was assumed. Chest radiograph showed mildly increased infiltration over both hilar areas. Oropharyngeal mucous membranes and perioral skin were hyperemic and edematous.

The hemogram showed leukocytosis, with a white blood cell count of 23,200 cells/mm³. Blood sugar was 295 mg/dL and venous blood gas analysis showed a pH of 7.27, PaCO₂ 35.5 mmHg, and HCO₃⁻ 16.1 mmol/L. Serum alanine aminotransferase was 21 U/L and creatinine was 0.5 mg/dL. Results of the electrocardiogram were normal. Dopamine and dobutamine were continuously infused. Ampicillin-sulbactam was administered to treat the aspiration pneumonia.

On the 2nd hospital day, the patient regained consciousness and inotropic agents were tapered. Generalized exanthema was found over his entire body. White plaques and sloughing of the oropharyngeal mucosa were observed. Oropharyngeal and esophageal burn were highly suspected. Esophagogastrosocopy was performed and revealed diffuse white plaques, ulcerations, and sloughing of the mucosa in a circumferential pattern over the whole esophagus, with concomitant hemorrhagic gastritis (Figure).

Intravenous corticosteroid (hydrocortisone 40 mg every 6 hours) and H2 blocker were administered for 3 days. A nasogastric tube was inserted as an intraluminal stent. Total parenteral nutrition was instituted starting on the 3rd hospital day, but hypertransaminasesemia developed from the 15th hospital day. The infant began feeding via a nasogastric tube from the 15th hospital day, and serum aminotransferases gradually returned to normal. He resumed oral intake with infant formula from the 23rd hospital day, and the nasogastric tube was removed on the 28th hospital day. The infant was discharged on the 30th hospital day. Esophagogram was suggested at 1 month after discharge, but was refused by his parents. His oral intake remained well without dysphagia at 1 year after discharge even while eating solid food. There were no overt neurologic or visual deficits at 22 months after discharge.

**Discussion**

Chloral hydrate is widely used in pediatric patients undergoing diagnostic procedures. In children, a single dose of 50–100 mg/kg of body weight produces adequate sedation. It is generally considered to be a safe and effective drug in infants and children.³ Adverse effects, such as central nervous system depression and cardiac arrhythmia, have been reported in some children.²

Gastrointestinal irritation is a well-known side effect of chloral hydrate administration, but it is usually mild. The irritant actions of chloral hydrate give rise to an unpleasant taste, epigastric discomfort, nausea, and occasional vomiting. Severe irritations to the gastrointestinal tract after chloral hydrate overdose are rarely reported. A 66-year-old female orally ingested 18 g of chloral hydrate in a suicide attempt. Four months later, she developed esophageal stricture requiring esophageal dilatation.³ A 25-year-old female developed gas-

![Figure](image-url)
tric necrosis 4 days after ingesting 20 g of chloral hydrate. She was treated with the Roux-en-Y procedure to bypass the stricture at the esophagogastric anastomosis. A 68-year-old woman ingested 10 g of chloral hydrate in a suicide attempt; gastric perforation was found immediately after the chloral hydrate ingestion.

The pathogenesis of esophageal burn secondary to chloral hydrate ingestion is unclear. The corrosive effects of chloral hydrate may be increased by insufficient dilution and overdose. The chloral hydrate should be diluted to a concentration of 10%. In our patient, the 8 g of chloral hydrate was diluted with only 15 mL water (53%, pH 3.45). Because the patient developed generalized exanthema over his entire body, whether he exhibited an idiosyncratic reaction to chloral hydrate is another consideration. In our patient, the esophagogastroscopic examination revealed severe esophageal burn with diffuse sloughing of the mucosa along the whole esophagus. Generally, the more severe the esophageal burn, the higher the likelihood of subsequent stricture formation. As the efficacy of steroid treatment in preventing stricture is controversial, and increased vulnerability to infection is a concern, we used a short-term course of steroids for controlling any possible hypersensitivity reaction. Since the natural course of chloral hydrate-induced esophageal burn is unknown, it is also difficult to determine how long the nasogastric tube should be inserted, and whether or not steroid use is beneficial.

In conclusion, this report illustrates the need for awareness among pediatricians that chloral hydrate overdose and inadequate dilution with water in the sedation of children for diagnostic procedures can lead to the severe complication of esophageal and oropharyngeal burn. After chloral hydrate ingestion, the infant or child should be assessed for the presence of any oropharyngeal burn. Early recognition is essential in the management of this complication.

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References