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Case Report

An autopsy case of zinc chloride poisoning

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ABSTRACT

Ingestion of large amounts of zinc chloride causes corrosive gastroenteritis with vomiting, abdominal pain, and diarrhea. Some individuals experience shock after ingesting large amounts of zinc chloride, resulting in fatality. Here, we present the results of an administrative autopsy performed on a 70-year-old man who ingested zinc chloride solution and died. After drinking the solution, he developed vomiting, abdominal pain, and diarrhea, and called for an ambulance. Except for tachycardia, his vital signs were stable at presentation. However, he developed hypotension and severe metabolic acidosis and died. The patient's blood zinc concentration on arrival was high at 3030 μg/dL. Liver cirrhosis with cloudy yellow ascites was observed, however, there were no clear findings of gastrointestinal perforation. The gastric mucosa was gray–brown, with sclerosis present in all gastric wall layers. Zinc staining was strongly positive in all layers. There was almost no postmortem degeneration of the gastric mucosal epithelium, and hypercontracture of the smooth muscle layer was observed. Measurement of the zinc concentration in the organs revealed the highest concentration in the gastric mucosa, followed by the pancreas and spleen. Clinically, corrosive gastroenteritis was the cause of death. However, although autopsy revealed solidification in the esophagus and gastric mucosa, there were no findings in the small or large intestine. Therefore, metabolic acidosis resulting from organ damage was the direct cause of death.

1. Introduction

Zinc chloride is very hygroscopic. When dissolved in water, it forms hydrogen chloride and the highly corrosive zinc oxychloride. Ingestion of large amounts of acid causes corrosive gastroenteritis with vomiting, abdominal pain, and diarrhea resulting from the stimulatory action on the mucous membranes. Some individuals experience shock after ingesting large amounts of zinc chloride, resulting in fatality [1–6]. Here we present the results of an administrative autopsy performed on a man who ingested zinc chloride solution and died approximately 11.5 h later.

2. Case report

A 70-year-old man presented at the hospital after ingesting 250 mL of zinc chloride solution (concentration unknown), which he used at home for soldering. After drinking the solution, the subject developed vomiting, abdominal pain, and diarrhea, and called for an ambulance. Although he had intended to commit suicide, he told emergency medical workers that he mistook the solution for mouthwash.

Immediately after arrival at the hospital, the patient's vital signs were stable except for tachycardia. However, 2 h later he developed hypotension. Despite massive fluid administration (>10 L of Ringer's solution) and administration of chelating agents (ethylenediaminetetraacetic acid calcium disodium salt hydrate: Ca-EDTA), maintenance of blood pressure was difficult. Severe metabolic acidosis developed, for which continuous hemodiafiltration was performed. However, the acidosis did not improve and the subject's respiratory condition deteriorated. Approximately 10 h after arriving at the hospital, the subject died. The patient's blood

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zinc concentration on arrival was high at 3030 μg/dL (normal blood concentration is 65–110 μg/dL).

2.1. Autopsy findings

Marked generalized edema, assumed to have resulted from massive fluid administration, was observed. Liver cirrhosis and splenomegaly were observed, with approximately 1300 mL of cloudy yellow ascites. However, there were no clear findings of gastrointestinal perforation. The pharyngeal and esophageal mucosa was light gray. The gastric wall revealed rigid degeneration (Fig. 1A). The gastric mucosa was gray-brown, with sclerosis present in all gastric wall layers. The corrosive changes resulting from zinc did not extend beyond the stomach (Fig. 1B). The stomach contained 45 mL of yellow fluid. Mild intestinal edema was observed; however, there were no pathological findings in the mucosa of the small or large intestine. The femoral vein blood alcohol concentration was 0.00 mg/mL, and drug screening with Instant-View M-1™ (Alfa Scientific Designs Inc., Poway, CA, USA) was negative.

2.2. Histological findings

Compared with ordinary autopsy specimens, there was almost no postmortem degeneration of the gastric mucosal epithelium, and there was marked congestion in the submucosa (Fig. 2A). A characteristic finding was excessive contraction of the smooth muscle layer of the muscularis propria (Fig. 2B). There were no remarkable histological changes in the esophageal mucosa. The mucosal epithelium of the duodenum showed normal postmortem changes. There were no other pathological findings in the mucosa of the small or large intestine. Congestive edema was observed in the lungs. Although fibrosis of the myocardium was observed, there was no myocardial necrosis. Previous reports have described renal tubular damage in acute zinc poisoning; however, only arteriosclerotic renal atrophy was found in this case, with no marked changes in the renal tubules.

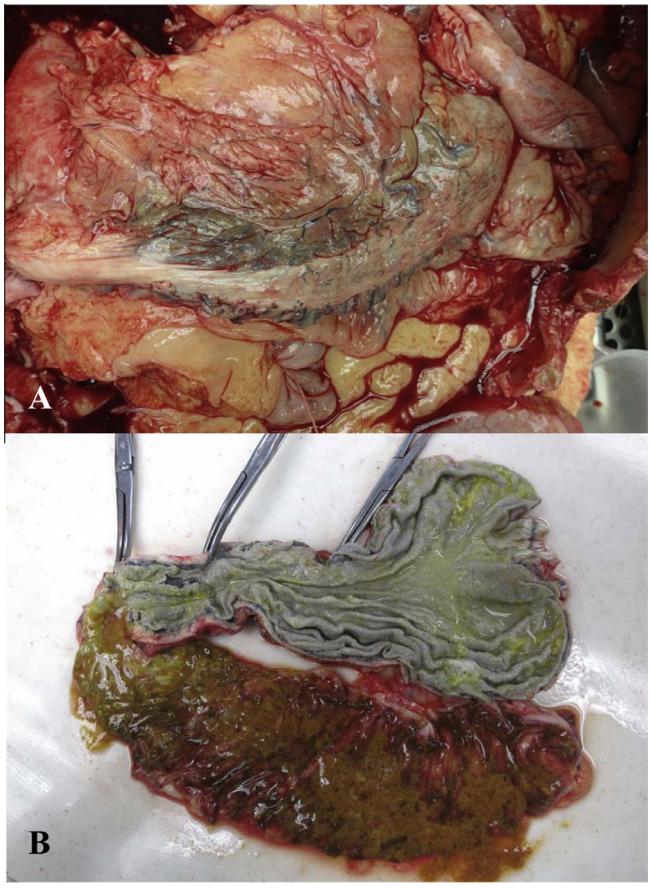

Fig. 1. Macroscopic images of the stomach and duodenum. A. Anterior surface of the stomach. The gastric wall shows rigid degeneration, with sclerosis through all layers. B. Mucosal appearance of the stomach and duodenum. The entire gastric mucosa is gray-brown. The corrosive changes caused by zinc chloride are limited to the stomach. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

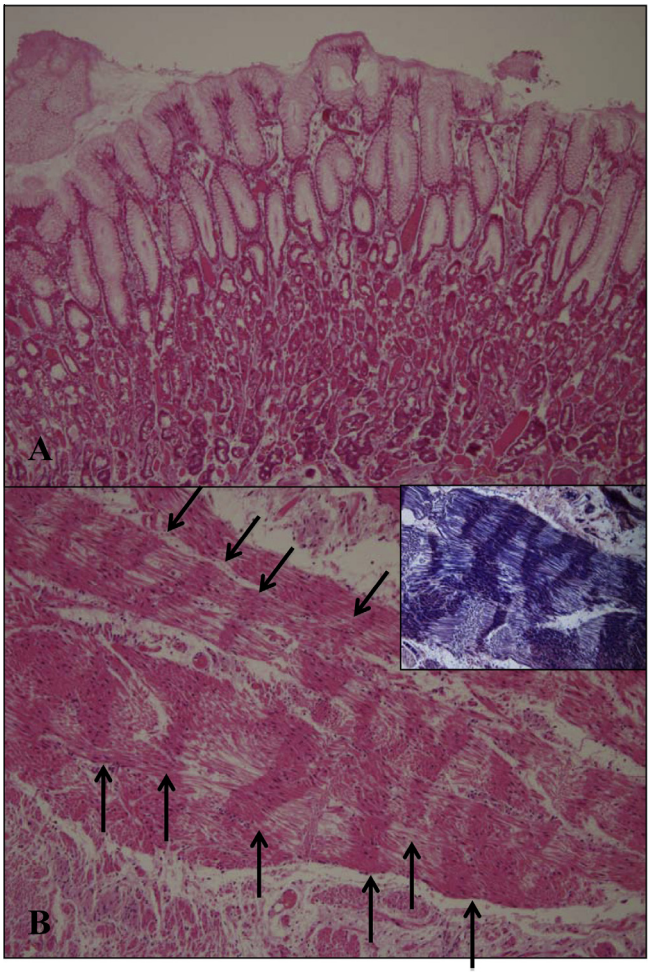

Fig. 2. Histological images of the gastric mucosa. A. Mucosal layer and lamina propria mucosae. Compared with ordinary autopsy specimens, almost no postmortem degeneration of the gastric mucosal epithelium is observed. B. Muscularis propria layer. Hypercontracture of the smooth muscle layer is observed (inset: phosphotungstic acid staining).

To evaluate zinc distribution in the gastrointestinal tract, we performed histochemical staining specific for zinc (dithizone [diphenylthiocarbazone] staining) [7]. One milligram of dithizone (Nacalai Tesque, Kyoto, Japan) was dissolved with 1 mL DMSO (dimethylsulfoxide), one drop NaOH, and 30 mL distilled water. The specimens were stained with dithizone solution for 10 min, and counterstained with hematoxylin. All layers of the gastric mucosa were strongly positive for zinc (Fig. 3A). In contrast, approximately half of the esophageal epithelium was only mildly positive, and the duodenum was negative (Fig. 3BC).

2.3. Zinc concentrations

Liquid specimens had the following zinc concentrations: femoral vein, 1883 μg/dL (3030 μg/dL at emergency room); urine, 37 μg/dL; cerebrospinal fluid, 95 μg/dL.