The Journal of Emergency Medicine, Vol. 40, No. 3, pp. 276–279, 2011

0736-4679/$–see front matter

doi:10.1016/j.jemermed.2007.12.030

Clinical Communications: Adults

CATASTROPHIC GASTROINTESTINAL INJURY DUE TO BATTERY ACID INGESTION

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□ **Abstract—Background: Acids account for 20% of all chemical exposures through various routes. Caustic acids such as hydrochloric and sulfuric acid are common ingredients in many household and industrial products. Due to the corrosive properties of these substances, tissue injury caused by oral exposure can lead to severe esophageal and gastrointestinal burns. Case Report: We report a case of a patient presenting with severe acidosis, who required multiple laparoscopic evaluations to assess various gastrointestinal tract injuries and who ultimately underwent total gastrectomy. The diagnosis was made primarily based on the arterial blood gas and esophagogastroduodenoscopy findings, as well as the pathological examinations of various biopsied and resected tissues showing hemorrhagic necrosis of the esophagus, stomach, and small bowel. This patient eventually admitted to having ingested an unspecified amount of battery acid. Conclusions: Collaborative efforts by Emergency Medicine, Pathology, and General Surgery services are required for timely diagnosis, treatment, and management of patients after caustic acid exposures.** © 2011 Elsevier Inc.

□ **Keywords—battery acid; caustic ingestion; gastrectomy; acidosis; hemorrhagic necrosis**

INTRODUCTION

Caustic substances are common ingredients in industrial and household products as well as various cosmetic and personal care products. These items contain acids and alkalis such as hydrochloric and sulfuric acid, sodium and calcium hydroxide, and sodium hypochlorite (1). According to the most recent report by the American Association of Poison Control Centers (AAPCC), these products accounted for 18% of substances most frequently involved in adult exposures (2).

Ingestion of caustic acids (e.g., battery acid) can result in severe esophageal and gastrointestinal burns. Battery acid is sulfuric acid, which donates hydronium ions (H^+) to various substances, thereby disrupting covalent bonds, promoting tissue injury, and causing toxicity (1,3). The superficial mucosal tissue denaturation that takes place after acid ingestion is classically described as coagulation necrosis followed by eschar or protective coagulum layer formation. Due to the alkaline environment of the oropharynx and the ability of the esophagus to withstand exposure to gastric acid, these organs were previously thought to be somewhat protected from acid injury. However, despite rapid passage and penetration of H^+ after acid exposure, two studies have independently shown that 50% of hydrochloric and sulfuric acid ingestions resulted in severe esophageal injury and 55% of patients ingesting battery acid had confirmed esophageal injury (4,5). Furthermore, ingestion of more than 200 mL of battery acid in these patients leads to significant gastrointestinal burns (5). Thus, exposure to a high quantity of acid may produce full-thickness injury to the stomach,

RECEIVED: 22 May 2007; FINAL SUBMISSION RECEIVED: 7 December 2007;
ACCEPTED: 20 December 2007

Table 1. Selected Admission Laboratory Results

	Units	Reference Range	Patient
Respiratory			
pH	Units	7.400 ± 0.030	6.915
pCO_2	mm Hg	40.0 ± 3.0	24.8
pO_2	mm Hg	90 ± 10	129
HCO_3	mmol/L	22.0 ± 2.0	5.0
Base excess			−28
Hematology			
White blood cells	10^6	4.10–10.80	25.89
Red blood cells	10^6	4.37–5.74	4.92
Hemoglobin	gm/dL	13.7–17.5	14.6
Hematocrit	%	40.1–51.0	44.1
Platelets	10^6	140–370	285
Chemistry			
Sodium	mmol/L	138–147	142
Potassium	mmol/L	3.6–5.0	4.6
Chloride	mmol/L	100–108	104
CO_2	mmol/L	22–30	10
Glucose	mg/dL	65–110	256
Blood urea nitrogen	mg/dL	7–20	13
Creatinine	mg/dL	0.8–1.5	1.1
Calcium	mg/dL	8.4–10.2	9.5
Total protein	g/dL	6.3–8.2	9.0
Albumin	g/dL	3.9–5.0	4.9
Amylase	U/L	30–110	120
Lipase	U/L	25–300	392
Aspartate aminotransferase	U/L	10–47	37
Alanine aminotransferase	U/L	20–70	5
Anion gap		6–12	28

small bowel, and duodenum, even though the stomach is physiologically an acidic environment.

Although intentional battery acid ingestion is rare, it has significant morbidity and mortality. Rapid diagnosis and appropriate clinical management is critical (6–9). We report a case of hemorrhagic necrosis of the esophagus, stomach, and small bowel secondary to caustic ingestion of battery acid.

CASE REPORT

A 54-year-old man presented to the Emergency Department (ED) complaining of intolerable abdominal pain and nausea that began 1 h earlier. Despite prior history, he denied intentional self-harm. Upon evaluation, the patient exhibited mild abdominal distension, tenderness, and was in acute distress. His vital signs were: blood pressure 164/87 mm Hg, heart rate 87 beats/min, and respiratory rate 26 breaths/min. Shortly after arrival and evaluation, the patient was found to be in extremis and was intubated. During the intubation, the posterior oropharynx appeared abnormal; the oral mucosa was white to pale grey in color. Given the patient's history, presenting symptoms, and subsequent rapid decline, ingestion of a caustic substance was suspected. After aggressive stabilization, a central line was placed and blood was collected for arterial blood gases (ABG), comprehensive metabolic panel (CMP), complete blood count (CBC), and toxicology screen.

The ABG revealed a severe anion gap acidosis (Table 1). Serum toxicology and volatiles screens were negative. The CBC, CMP, amylase, and lipase were within normal limits (Table 1); however, the white blood cell count and serum bicarbonate were severely abnormal. Due to concern for ischemic bowel, a computed tomography (CT) scan of the abdomen was obtained. It demonstrated a thickened stomach and small intestine (Figure 1). Despite the patient's denial of suicidal ideation, the oropharyngeal burns, laboratory and radiographic results were all felt to be consistent with ingestion of a caustic liquid.

To determine the cause of rapid patient decompensation and survey gastrointestinal integrity, an emergent exploratory laparoscopy was indicated and performed within 2 h of admission. Approximately 300 cm^3 of hemorrhagic ascites was removed. Full-thickness necroses of the stomach (from the gastroesophageal junction through the antrum) and all four portions of the duodenum were present. Gross examination of the small bowel segment demonstrated brown-black, soft, friable and ischemic mucosa along the entire surface, including the resected margins. In addition, focal discoloration (gray to black-tan) was evident at the serosal surface. Histology confirmed transmural necrosis of the small bowel.

The catastrophic gastrointestinal necrosis and presence of posterior oropharyngeal burns were consistent with the suspicion of caustic ingestion. Upon closure of the abdomen, an esophagogastroduodenoscopy (EGD)

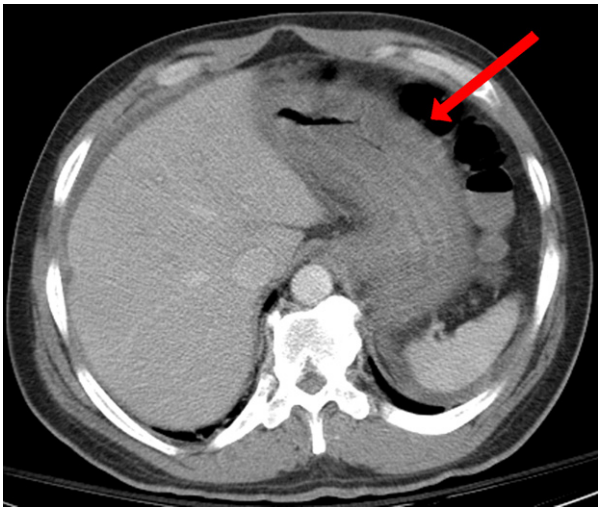

Figure 1. Computed tomography scan of the abdomen at admission demonstrating markedly thickened gastric wall involving the entire stomach.