

# The Dire yet Regrettable Cause of Upper Gastrointestinal Bleeding: Caustic Substance Ingestion

Ana L. Romero<sup>1</sup>, Jesus Romero<sup>1\*</sup>, Sherif Elkattawy<sup>2</sup>, Nikhita Sachdeva<sup>3</sup>, Fareeha Abid<sup>1</sup>, Hardik Fichadiya<sup>1</sup>, Muhammad Atif Masood Noori<sup>1</sup>, Islam Younes<sup>1</sup>, Shruti Jesani<sup>1</sup>, Ricardo Rodriguez<sup>1</sup>

<sup>1</sup>Internal Medicine Department, RWJBarnabas Health/Trinitas Regional Medical Center, Elizabeth, NJ, USA.

<sup>2</sup>Cardiology Department, St. Joseph's University Medical Center, Paterson, NJ, USA.

<sup>3</sup>Internal Medicine Department, St. George University, Grenada.

\*Corresponding author: je-romeros@hotmail.com

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**Abstract** Caustic ingestion results in a wide range of injuries from mucosal erythema to transmural necrosis and viscus perforation, depending on the nature, amount, and time of contact of the substance with the gastrointestinal mucosa. Esophagogastroduodenoscopy (EGD) is considered the gold standard not only for the diagnosis but also for the guide in management. Here in we report a case of a 44-year-old male who presented to ER with complaints of abdominal pain and coffee ground emesis after ingestion of an acid cleaner in the setting of a suicide attempt. CT chest and abdomen demonstrated a small amount of fluid around the distal esophagus and diffuse gastric submucosal edema with no pneumo-mediastinum. EGD showed severe caustic esophagitis and necrosis, gastritis, and mucosal necrosis. Gradually patient transitioned from parenteral nutrition to a liquid diet. Given patient continued to have symptoms of dysphagia, a follow-up EGD was performed which showed erosive esophagitis and severe intrinsic stenosis 26.5 cm from the incisors. The patient underwent gastrostomy as an attempt failed to traverse the stenosis. Post-procedure, the patient started spiking fever with leukocytosis and was found to have an intra-abdominal abscess as evidenced by CT abdomen for which he had emergent laparotomy and evacuation of abdominal abscess and replacement of gastrostomy tube. This case represents the complicated nature of ingesting acidic fluid. It also highlights the importance of considering of overall worse prognosis with that of alkali as compared to acid.

**Keywords:** *gastrostomy, esophagogastroduodenoscopy, intra-abdominal abscess, esophageal stricture, caustic acid ingestion*

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## 1. Case Presentation

This is a 44-year-old Hispanic male patient with no significant past medical history, who was brought to the Emergency Department at Trinitas Regional Medical Center on November 11, 2021, for complaining of abdominal pain and coffee-ground emesis after ingesting Sure Klean 600, an acidic cleaner that he uses at his job as a construction worker, as a suicidal attempt. As per Emergency Medical Service, the patient was found by his neighbor on the ground in front of his house with an episode of dark bloody emesis.

During assessment at the Emergency Department, the patient had a coffee-ground emesis episode, and he was found to be agitated and uncooperative, however, no active drooling or stridor was evidenced. Physical examination was unremarkable. At that time, the patient was intubated and sedated with Midazolam for airway

protection. Of note, during intubation, no laryngeal edema or active bleeding was evidenced. Hence, the patient was admitted to ICU for further evaluation and management.

Upon admission, blood work revealed a WBC of 23.6 [4.8-10.8 K/UL], and a hemoglobin level of 16 [14-18 GM/DL]. ABG showed a pH of 7.22 [7.35-7.45], pCO<sub>2</sub> of 24 [35-45 mmHg], and HCO<sub>3</sub> of 12.5 [22-26 mmol/L]. The lactic acid level was 2.1 [0.5-2.2 mmol/L]. UDS was positive for tricyclic antidepressants and benzodiazepines. CT scan of the chest, abdomen, and pelvis with contrast showed a small amount of fluid adjacent to the distal esophagus likely corrosive injury with no pneumomediastinum, diffuse gastric submucosal edema with no pneumoperitoneum.

Poison control was contacted, and they recommended follow-up arterial blood gases, lactic acid, basic metabolic panel, and giving bicarbonate ampullas. Recommendations were followed and the patient was also started on IV pantoprazole infusion and piperacillin-

tazobactam in the setting of leukocytosis, possibly secondary to aspiration.

During ICU stays, arterial blood gases and lactic acid improved, and the basic metabolic panel remained within normal limits. An upper GI endoscopy was performed on November 15, 2021, which revealed severe caustic esophagitis and necrosis, gastritis, and mucosal necrosis. Since leukocytosis improved and there was no finding consistent with perforation on EGD, the antibiotic was discontinued. In the need of starting total parenteral nutrition, a PICC line was placed by Interventional Radiologist. He was successfully extubated on November 16, 2021, and since he remained hemodynamically stable, he was transferred to the medical floor.

Once on the medical floor, the patient was progressively transitioned from TPN to a liquid full diet and to dysphagia pureed level 1 while ongoing TPN. However, at that time, the patient started to complain of odynophagia and dysphagia hence modified barium swallow was performed and it showed findings consistent with esophageal spasm. A follow-up EGD was performed on December 7, 2021, which showed erosive esophagitis and an intrinsic severe stenosis measuring 7 mm (inner diameter) which was found at 26.5 cm from the incisors. Attempt to traverse the stenosis with gentle pressure failed, hence surgery was consulted for feeding tube placement.

On December 12, 2021, the patient was taken to the Operating Room for Stamm gastrostomy. Three days following the procedure, the patient spiked low-grade fever with Temp 100.2 F, and with leukocytosis up to 14,000 [4.8-10.8 K/UL]. On physical examination, the midline incision had purulent brown and foul-smelling drainage. At that time, wound cultures were sent, and the patient was empirically started on piperacillin-tazobactam 3.375 mg three times a day.

The following days, the patient's hospital course was characterized by the presence of findings consistent with pneumoperitoneum and intraperitoneal abscess evidenced on CT scan with contrast of abdomen and pelvis, which prompted emergent laparotomy with abscess evacuation, and replacement of gastrostomy tube. However, the patient remained persistently tachycardic, with low-grade fever with a Temp of 100.4 F, and new findings consistent with multiple loculated ascites at the liver, perihepatic space, with additional fluid by the spleen in the left upper quadrant were evidenced.

Wound culture showed lactose fermenting gram-negative rods and blood cultures grew *Klebsiella*. At that time, antibiotic coverage was broad to meropenem 1 g three times a day, which was subsequently de-escalated to ceftriaxone 2 grams daily. On December 23, 2021, a follow-up CT scan of the abdomen showed small loculated perihepatic ascites, some intra-peritoneal free air with scattered postoperative fluid collections, somewhat improved from before. At that time, Interventional Radiologist was consulted for possible drainage of fluid collections; however, no surgical intervention was indicated.

Over the following days, the patient was continued on meropenem, with significant subjective and objective improvement. He was discharged with 7 more days of trimethoprim/sulfamethoxazole and with follow-up as an

outpatient with surgery, wound care, and internal medicine.

## 2. Discussion

Hydrochloric acid is often used as an additive in cleaning products because of its chemical properties that neutralize alkaline agents. Despite the benefits it provides, there is an increased risk for acid ingestion by individuals due to its presence in common household cleaning products. While ingestion of corrosive acid in children is usually accidental, ingestion in adults is almost always associated with suicidal attempts. The extent of the injury is dependent on the volume, concentration, commercial preparation, and mode of ingestion of the acid [1]. The mortality rate due to accidental acid ingestion is around 20% but that rate increases to 78% within the context of suicide attempts [2]. Knowing how to treat a patient that has ingested corrosive acid requires an understanding of the physiological processes that lead to symptoms, both present and hidden. Acids can induce coagulative necrosis via protein modification and risk perforating the gastrointestinal system [3].

Most symptomatic patients who have ingested corrosive acids are evaluated based on their clinical presentation followed by fiberoptic endoscopy <12 hours after ingestion [4]. Common acute signs and symptoms include nausea, vomiting, drooling of saliva, dysphagia, retrosternal chest pain, and dyspnea [5]. Late clinical findings may include esophageal strictures and stenosis, gastrointestinal reflux, esophageal carcinoma (potentially manifest years later), and stenosis of gastric antrum/pylorus [6]. Secondary bacterial infections may lead to mediastinitis and perforation, gastritis, stomatitis, gingivitis, pneumonia, and peritonitis [7]. These infections can increase the mortality rate by five-fold [8]. Our patient was administered piperacillin-tazobactam secondary to aspiration as a precautionary measure but due to his utmost hospital care, no respiratory or intrathoracic infection ensued. Dr. Alfredo Rossi, MD established a 6-step management course for acid ingestion in patients. Assess vital signs (including pH, CMP, BMP, ECG, and X-ray). Leukocytosis and metabolic acidosis are the most common biological signs of severity [9]. Analyze symptoms and correlate complications: for example, abdominal pain (perforation), dysphagia (airway involvement), hematemesis (extensive/severe injury), etc. Detection of objective signs. Identification of substance. Characteristic of ingestion (modality and amount). Radiological evaluation. Each patient will receive a thoracoabdominal X-ray to look for signs of perforation, pneumomediastinum, or pneumonia.

Endoscopy should always be ordered for patients with suicidal ingestion of caustic/corrosive substances as 70% of these patients have no oral pathological findings [10]. On account of how fast the patient swallowed the corrosive material, intraoral irritation may not be visible, while intestinal damage may be life-threatening. CT and gastrografin are reserved for more in-depth suspicions [11]. One should never induce vomiting in a patient as the second contact of acid on the esophageal mucosa can increase transmural tissue damage. The nasogastric tube

should not be blindly placed as it is a risk for perforation. The utmost priority is to establish airway management to avoid edema restricting the airways. Our patient was triaged, intubated, and transferred to the ICU immediately as these 6 steps were executed efficiently.

On endoscopy, caustic lesions can be classified via the Zargar classification system. The ranking is as follows: Grade 0 (normal), Grade 1 (mucosal edema and erythema), Grade 2A (superficial ulcers, hemorrhage, erosions, blisters), Grade 2B (circumferential lesions), Grade 3A (Focal deep gray/brown/black ulcers: necrosis), Grade 3B (extensive 3A), and Grade 4 (perforations) [12].

There are no therapeutic criteria for acid ingestion; however, treatment with PPI, steroids, antibiotics, endoscopic dilation, and surgical intervention are all centered around avoiding esophageal strictures and improving patient care. Total parenteral nutrition should be slowly weaned to a full liquid diet or as the patient tolerates it. A close eye should be kept on the patient's electrolytes, acid-base status, and blood count. Our patient's metabolic acidosis and leukocytosis decreased during their ICU stay. No surgical intervention was needed as the endoscopy showed low-grade esophagitis and necrosis. All in all, early therapeutic intervention, admission to ICU, and endoscopic evaluation are necessary for the management of corrosive substance ingestion.

Even though alkali substance ingestion tends to cause worsened esophageal damage, acidic substances can also result in life-threatening damage. Esophageal strictures usually develop within 2 months, although can be evidenced as early as two weeks after esophageal damage. Early endoscopy is recommended however it can be delayed in the setting of hemodynamic instability. Neutralizing agents are not recommended due to the risk

of additional injury due to the risk of an exothermic reaction.

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