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Catastrophic Upper Gastrointestinal Tract Complications Following Corrosive Ingestion

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ABSTRACT

Ingestion of caustic substances, either acidic or alkali, is a well-known cause of significant mortality and morbidity in the pediatric and adult populations. According to the pH, physical form, amount, and rate of ingestion of these substances, a wide range of immediate as well as possible subsequent chronic complications can result. Some patients may have minimal to mild symptoms. However, others can present with moderate or even serious symptoms, in the form of dysphagia, odynophagia, hoarseness, and epigastric pain. Herein we present a case of a 26-year-old male patient with a history of Nitric acid ingestion. He initially complained of severe chest and abdominal pain, associated with hematemesis, dysphagia, odynophagia, hypersalivation, and inability to tolerate oral intake. Initial Esophagogastroduodenoscopy (EGD) showed diffuse mucosal ulceration reaching the second part of the duodenum. Multiple subsequent EGDs revealed more extensive damage and subsequent esophageal stricture and pyloric stenosis for which multiple dilatation attempts were done. The patient received extensive supportive management during his hospitalization course, but the damage was severe enough that he was referred for surgical management. Such catastrophic sequels of caustic ingestion are still encountered, especially in developing countries. We present this case looking forward to raising awareness about this dangerous phenomenon and highlighting the significance of immediate identification and grading of the injury.

Keywords: Caustic injuries, esophageal Stricture, pyloric stenosis, Corrosive

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Introduction:

Although rare in developed countries, corrosive ingestion is a serious public health problem that developing countries still suffer from due to a lack of strict regulatory measures upon causative agents. Generally, for a substance to be notorious for causing corrosive injury to tissues, it must be either an acid with a pH less than three or a base with a pH greater than 11[1]. Most corrosives are found in cleaners, detergents, vinegar, hair relaxers, and disk batteries. Commonly ingested corrosive materials include nitric acid, phosphoric acid, acetic acid, hydrochloric acid, and sodium hydroxide [2].

Two age groups are most at risk for caustic ingestion: children (80% of the cases) who ingest corrosives unintentionally with mild injuries mostly, and adults who usually ingest strong corrosives with suicidal intent [3, 4]. Adult suicidal ingestion tends to harm the oral cavity, oropharynx, and proximal part of the esophagus because suicide is challenging, and patients may be reluctant at first. On the other hand, accidental ingestion has been shown to cause more rapid, substantial, and thus more distal injuries to the gastrointestinal tract (GI) [5, 6].

The clinical presentation shows diversity depending on the extent and severity of the injury. Most patients are mild asymptomatic to slightly symptomatic, e.g., throat pain with or without erythema. However, moderately to severely injured patients are significantly symptomatic, affecting the lips, oral cavity, and pharynx [5]. The most prominent symptoms are odynophagia, dysphagia, hoarseness, and epigastric pain. Life-threatening complications like massive hemorrhage, gastrointestinal tract perforation, and aorto-enteric fistula may also occur [7]. Moreover, when the patients overcome these short-term complications, they may develop long-term sequelae including esophageal strictures (ES), tracheoesophageal fistula, tracheal stenosis, gastric outlet obstructions (GOO) or squamous cell carcinoma of the esophagus [8].

Herein, we present a case of a 26-year-old male patient who presented to our hospital after accidental ingestion of acidic substance with massive GI involvement. We will discuss laboratory, radiological, and endoscopic investigations during the hospital stay.

Case presentation:

A 26-year-old male from Gazza was referred to the gastroenterology center three weeks after accidental ingestion of 250ml of nitric acid. Other than smoking, the patient has negative medical and psychiatric history. At his initial hospital presentation, the patient was complaining of severe diffuse chest and abdominal pain that was continuous, progressive, and stabbing in nature. The pain was associated with hematemesis, dysphagia for both solids and liquids,odynophagia, and hypersalivation. The patient was admitted to Al-Shifaa hospital and managed conservatively with intravenous (IV) fluids, antibiotics, and analgesics for two weeks. Examination of oral cavity and oropharynx at that time was normal. During the last days, the symptoms' severity continued to increase with multiple episodes of vomiting of small amounts of gastric content, during which the patient required ICU admission for 4 days. After then, the attending gastroenterologist decided to perform an Esophagogastroduodenoscopy (EGD), which revealed diffuse mucosal ulceration throughout upper GI tracts reaching the second part of the duodenum. Accordingly, they decided to refer the patient to our hospital for advanced follow up and management.

On admission, physical examination revealed hemodynamically stable adult that looked tired and in pain. The patient was still complaining of the same symptoms he was referred with, as well as multiple episodes of melena that began two days prior to admission. He has had no oral intake since the incident expect for small amounts of water and milk. In the initial evaluation of the patient, his laboratories showed normal liver function tests (LFTs), kidney function tests (KFTs), serum electrolytes, complete blood count (CBC) except for drop in

hemoglobin (10 mg/dl). He was started on IV fluids, antibiotics, high-dose IV proton pump inhibitors (PPI), high protein nutrition (ENSURE), and opioid analgesics (pethidine) regularly.

Diagnostic EGD was performed within 48 hours and demonstrated severe esophagitis with stricture, severe inflammation at esophagogastric junction (EGJ), and severe gastropathy with antral stricture, Figure (1). Chest and abdominal computed tomography (CT) scan were done. The antral part of the stomach and proximal part of the duodenum appears collapsed with mural thickening surrounded by fat stranding correlating with endoscopic finding. No evidence of free air or fluid was noted in pleural or peritoneal cavities. According to a surgical consult, a jejunostomy feeding tube was planned and performed without a post-surgical complication. Nutrition was started with 2000 calories daily requirements, and instructions were given to increase the calories according to the patient tolerance.

During a period of seven weeks of hospital stay thereafter, the patient underwent six further endoscopic procedures with different diagnostic and therapeutic interventions shown in the table below (Table 1). During EGD 5, due to severe duodenal stricture, the duodenum perforated after the dilatation with extravasation of the contrast. The defect was closed immediately with four clips. Consequently, an abdominal CT-scan was performed, showing extensive pneumoperitoneum and pneumomediastinum visualized in the lower chest. The patient was kept NPO and managed conservatively with serial abdominal x-ray follow up until the duodenal perforation completely recovered.

After then, and despite all nutritional support, the patient continued to lose weight instead of gaining (catabolic state). Thus, the patient's case was discussed with our general, esophagus, and thoracic surgeons for surgical input. A decision was made for possible resection of the esophagus, stomach and proximal duodenum with colon transposition. This requires transferring the patient to an advanced center because that is not available in our center.

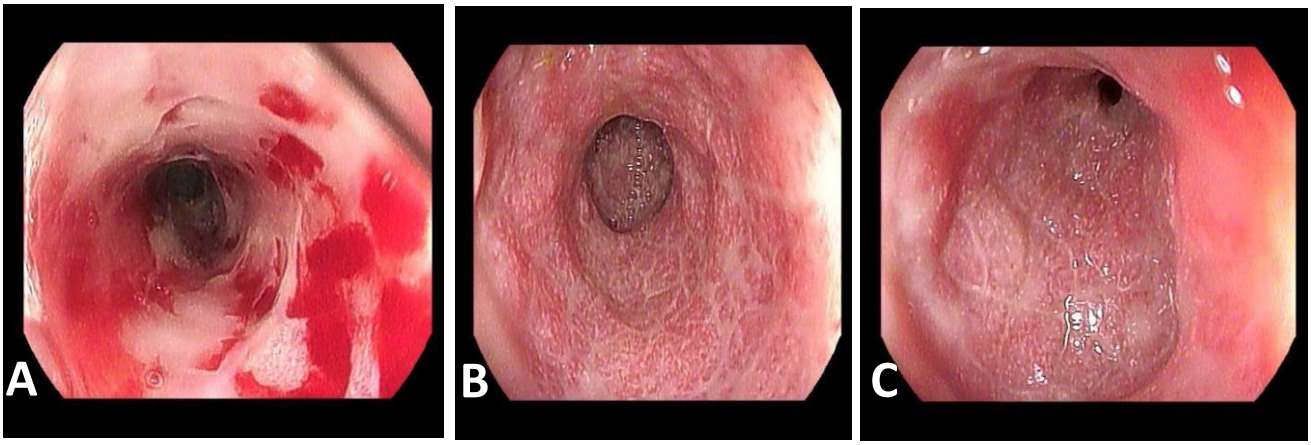

Figure 1: Esophagogastroduodenoscopy. A. Esophageal ulcerations, B. Antral stricture, and C. Pyloric stenosis (not reached).

Discussion:

Corrosive ingestion is a rare but potentially disastrous event, with long-term consequences that continue to affect developing countries such as Palestine due to the lack of government regulatory controls on causative agents [2, 9]. Oral Ingestion of Acids such as hydrochloric acid, acetic acid, and nitric acid leads to coagulative necrosis. Theoretically, this coagulative layer prevents further penetration into deeper tissues. Acids are thought to cause more gastric injury than esophageal injury because of the increased

esophageal transit time [1, 2, 10-13]. On the other hand, alkaline materials react with proteins, and fats produce liquefactive necrosis, increasing the possibility of deeper tissue penetration and transmural damage [11, 14].

Management of caustic UGI injuries aims to support the patient`s hemodynamics, assess the grade of injury, and prevent late complications [9]. Initial Clinical assessment of the patient alongside stabilization of the vitals and airway protection, intravenous fluid administration, and analgesics is considered the first step. Classic maneuvers such as vomiting induction, gastric lavage, and neutralization of alkali with acids and vice versa are no longer recommended because they aggravate the condition and lead to further damage to the esophagus from repeated exposure to the corrosive substance [10, 13, 15-18].

Table 1: Endoscopic findings, interventions, and complications during hospital stay

EGD Session	Day of admission	Findings	Interventions	Complications	Figure
EGD 2	11	Severe mucosal injury of the entire esophageal with stricture at 21 cm Severe gastric mucosal injury with stricuring Severe pyloric stenosis, not passed	TTS balloon dilatation to 8mm	Nil	Fig. 2: A&B
EGD 3	17	Esophageal ulceration and sloughed mucosa Severe gastritis with severe pyloric stenosis, not passed	Mid-esophagus and pyloric TTS balloon dilatation up to 10 mm	Nil	Fig. 2: C
EGD 4	23	Diffuse fibrotic changes of the esophagus and stomach Esophageal stricture at 28 cm Pyloric stenosis, not passed	TTS balloon dilatation to 10 mm of the esophageal stricture and pyloric stenosis	Nil	Fig. 2: D
EGD 5	27	Esophageal stricture Severe gastritis with luminal narrowing Severe pyloric stenosis Severe narrowing of the duodenal bulb	TTS balloon dilatation to 11mm of the esophageal stricture and pyloric stenosis	Duodenal perforation with contrast extravasation	Nil
EGD 6	34	Severe long esophageal stricture at 26 cm from incisors Severe gastric luminal narrowing with multiple pseudodiverticuli in the body Severe pyloric stenosis, not passed	TTS balloon dilatation up to 9mm of the esophageal stricture Fully covered biliary stent (10*100mm) deployed just below EGJ up to 26cm from incisors	Nil	Fig. 2: E &F
EGD 7	51	Two tight esophageal strictures at 17cm and 27cm Extensive gastric fibrosis, shrunken by scaring Tight pyloric stricture, not passed	Esophageal strictures dilatation to 10mm then to 12mm by CRE balloon over wire Previous esophageal stent removal	Nil	Fig. 2: G&H

EGD= Esophagogastroduodenoscopy, TTS= Through The Scope, EGJ= Esophagogastric Junction, CRE= Controlled Radial Expansion.

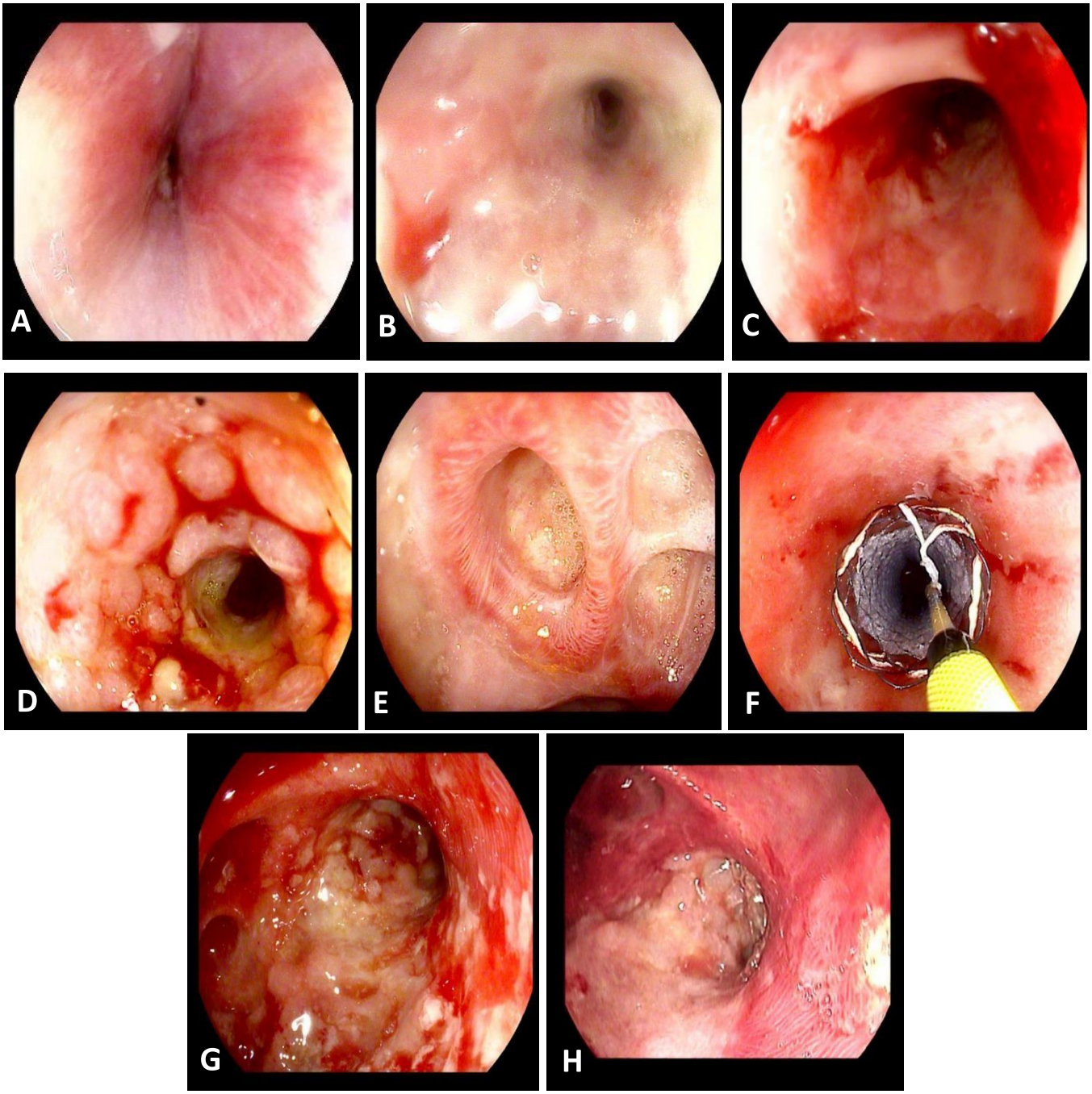

Figure 2: Esophagogastroduodenoscopy. A. Esophageal stricture at 21 cm, B. Severe pyloric stenosis, C. Pylorus post-dilatation, D. Esophageal tear and blood post-dilatation, E. Severe gastric narrowing with diverticula, F. Biliary stent in lower esophagus, G. Sever gastric scarring, and H. Gastric outlet obstruction.

To date, Esophagogastroduenoscopy (EGD) remains the cornerstone for diagnosing corrosive injuries worldwide. It helps determine the extent of the damage, predict morbidity and mortality, and thus helps guide subsequent management. Immediate endoscopy within 12 to 24 hours of caustic ingestion is the preferred method to provide grading for the injury [1, 3, 11, 19]. Several classification systems have been suggested for endoscopic findings in upper gastrointestinal corrosive injuries. Among them, the Zargar classification is widely accepted (Table2) [20]. Zargar studies' have revealed a significant association between the grade of mucosal injury and systemic complication, low-grade injuries (grades 1–2a) have a low risk of developing stricture, while strictures can occur in up to 80% of patients with high-grade injuries

(grades 3b). Also, patients with grade III injuries are more prone to early severe complications and death [3, 20, 21].

On the other hand, the main issue with EGD is the failure to estimate the depth of necrosis accurately. This may result in improper non-operative management adversely affecting survival and unjustified respective surgical procedure with long-term adverse consequences [10, 22, 23]. Furthermore, endoscopy is operator dependent with a high possibility of misinterpretation, especially when performed 24 hours later due to pathological changes that happened in the mucosa, like edema or bleeding [24].

Death and perforation are two short-term complications of caustic ingestion. Esophageal or stomach perforation can happen anytime during the first 2 to 3 weeks of ingestion; therefore, doctors should rule out these complications if deterioration or worsening of initially stable conditions happens [2, 25]. Late complications of caustic ingestion include gastric outlet obstruction, strictures, malignant transformation, and fistulation [10, 26]. Esophageal stricture is the most common and suffering late complication that's highly predictable in cases with Zargar grade 2b and 3a as the one in our patient. Typically, it starts to develop within 2-3 weeks post ingestion. Patients usually experience symptoms of dysphagia and substernal pressure. Gastric strictures are less common than esophageal strictures since the large diameter of the stomach occurs in 30-60% of individuals with Zargar scale 2b/3 injuries [11, 27, 28]. Our patient has concomitant esophageal and gastric strictures.

Table 2: Endoscopic classification of caustic injury – Zargar grading.

GRADE	ENDOSCOPIC FEATURES
GRADE 0	Normal mucosa
GRADE 1	Superficial mucosal edema and erythema
GRADE 2A	Superficial ulcerations, exudates, erosions
GRADE 2B	Deep discrete or circumferential ulcerations
GRADE 3A	Focal necrosis or brownish-black ulcers
GRADE 3B	Extensive necrosis
GRADE 4	Perforations

*This table is adopted from Contini and Scarpignato (2013) [2] and De Lusong et al. (2017) [11].

The first line of management is endoscopic dilation, which is much more effective earlier, while delayed initiation of dilation will increase the adverse events and decrease the success rate due to fibrosis and collagen deposition within the esophageal wall [10, 11, 13]. The duration between dilations ranges between one and three weeks, with three to five sessions required for satisfactory results [10]. While balloon dilation is the first management line, it has a high recurrence rate. In these conditions, an alternative option is intraluminal stenting insertion. Biodegradable, silicone rubber, and polyflex stents have shown promising results [2, 29, 30]. However, the failure of all previously mentioned conservative methods necessitates the surgical option of esophagectomy (partial or total) with colonic interposition or gastric pull-up [11].

Conclusion:

Caustic ingestion can lead to varying degrees of GI damage. After substance ingestion, a continuous damaging process can take place. This potentially can result in severe, diffuse, and extensive injuries that can still overwhelm

patients' lives, even years after the original damage. Hence, the main management priority is to hemodynamically stabilize the patient. It's widely accepted that EGD plays the main role in the diagnosis and estimation of the resultant injury-related morbidity and mortality, as well as guides to establishing the most appropriate case-dependent management plan. Thus, we reported this case in order to draw attention to such a dangerous scenario that continues to endanger much of society's manpower.

Ethical approval: The study is exempt from ethical approval in our institution.

Consent for publication: Written informed consent was obtained from the patient for publication of this report and accompanying images.

Declaration of competing interest: Authors declare no competing interest.

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