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Case Report

Endoscopic balloon dilatation in caustic-induced gastric outlet obstruction: case report

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ABSTRACT

The ingestion of caustic substances is a not infrequent problem that requires multidisciplinary, medical, surgical and endoscopic management, since it can cause minor burns to total necrosis of the digestive tract wall. A 76 years old male patient with a history of chronic alcoholism presented to the emergency department complaining of heartburn and abdominal pain after ingesting muriatic acid, accidentally mistaking it for alcohol. Computed tomography (CT) scan showed no signs of perforation and endoscopy revealed grade IIIB stomach burns. On day 45 a new endoscopy was performed, documenting fibrosis scars in the antrum with significant retraction and secondary pyloric stenosis, which was managed with endoscopic balloon dilatation (EBD) without complications. In this case, as in most patients with caustic-induced gastric outlet obstruction (GOO) can be successfully treated by EBD.

Keywords: Caustic ingestion, Caustic-induced gastric outlet obstruction, Endoscopy, Endoscopic balloon dilatation, Acid injuries, Alkali injuries

INTRODUCTION

There is a wide variety of solutions with corrosive properties, the most frequently identified caustic agents are found in cleaning products, bleaches, detergents, batteries, cosmetics, dyes, soaps, we can identify acids (hydrochloric, sulfuric, acetic acid) or alkalis (lye, caustic soda, lithium hydroxide).[1-3] The ingestion of caustic substances is a not infrequent problem that requires multidisciplinary, medical, surgical and endoscopic management, since it can cause minor burns to total necrosis of the digestive tract wall.[4] There are pH values that can be considered critical since they can produce important lesions with a pH less than 4 and greater than or equal to 12. Acid injuries include a series of processes triggered by protein coagulation and water loss with large areas of necrosis. Alkali injuries develop by tissue liquefaction, producing thermal burns due to hydration with great penetrating power (Figure 1).[5] However, pH alone does not explain why caustics cause different degrees of injury. The titratable acid or alkaline reserve (TAR) quantifies the amount of acid or base ion that the body's physiologic response donates to injured tissues to return them to physiologic pH.[6] Long-term sequelae are generally due to stenotic lesions, patients present with dysphagia due to enteric plexus injury and muscle tissue is replaced by scarring fibrosis.[7] In review studies it had been found that the most affected organs were the

stomach, esophagus and duodenum.[4] Worldwide epidemiological data were scarce mainly due to underreporting of caustic ingestion.[8] About 18,000 cases were reported annually in the United States 2 more than 75% were children under 5 years of age, followed in frequency by adults or adolescents from the population with a low socioeconomic level.[9] Clinical effects of caustic ingestions were divided into immediate, delayed and remote manifestations. The organ systems most involved were the eyes, skin, airway and gastrointestinal tract. Pain was often immediate, followed by loss of function. Common manifestations included swelling of the tongue and mouth, drooling and vomiting. Bleeding can be severe if the injury involved erosion of a vessel. Swelling of the airway caused stridor, respiratory compromise and changes in the voice. Perforation of the esophagus can lead to mediastinitis and perforation of the stomach or bowel can lead to peritonitis. In the latter case, perforation was often not initially accompanied by classic peritoneal findings on physical examination.[10]

CASE REPORT

A 76 year old male patient with a history of chronic alcoholism presented to the emergency department complaining of heartburn and abdominal pain after ingesting muriatic acid, accidentally mistaking it for alcohol. In the initial assessment, the patient was found to be algic with a tendency to tachycardia, abdomen without data of peritoneal irritation. CT scan of the chest and abdomen showed no signs of perforation and endoscopy revealed grade IIIB stomach burns (Figure 2). During the subsequent evolution, total parenteral nutrition and enteral nutrition by jejunostomy were started. On day 45, the patient started an oral diet and presented constant hyporexia, emesis and nausea. A new endoscopy was performed, documenting fibrosis scars in the antrum with significant retraction and secondary pyloric stenosis, which was managed with EBD without complications (Figure 3).

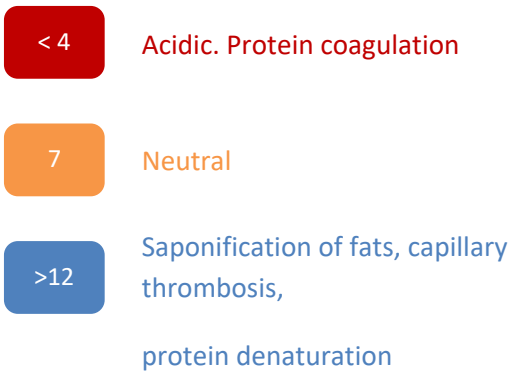

Figure 1: pH scale with corresponding acidic or alkaline values.

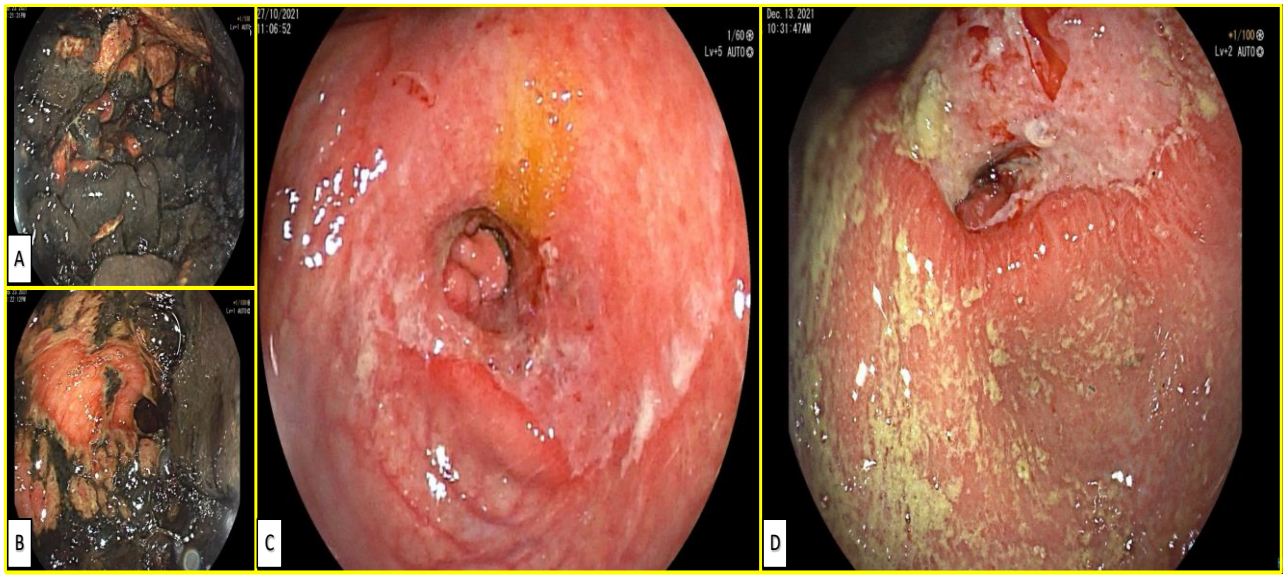

Figure 2: (A) Acute gastric necrosis, stomach; (B) necrosis in the antral and prepyloric region with irregular borders; (C) control endoscopy one month later, antrum with mucosal edema and hyperemia; (D) pylorus covered with fibrin, with stenosis that does not allow the passage of the endoscope.

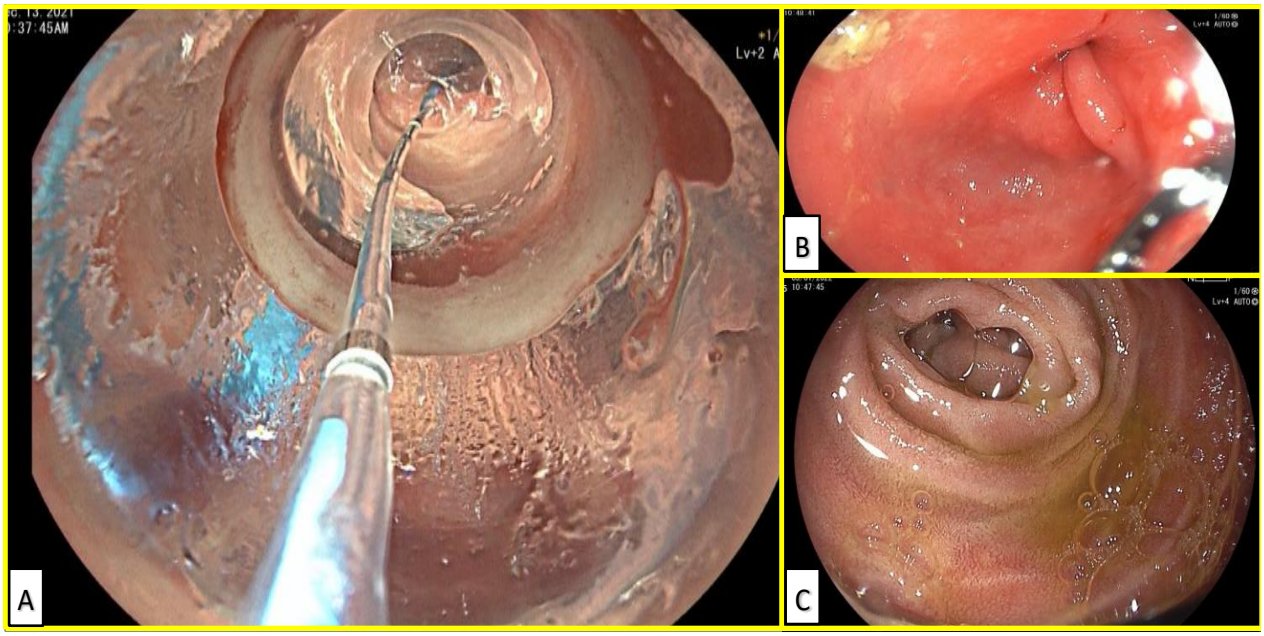

Figure 3: (A) Pneumatic dilation with balloon at 18, 19 and 20 mm; (B) antral mucosa with edema and permeable pylorus to the passage of the endoscope after dilation; (C) microscopically normal duodenal mucosa.

DISCUSSION

Due to the easy access to acid and alkaline products, the number of cases of accidental corrosive esophagitis and self-harm was increasing. The accidental ingestion of caustics usually affected mainly children and the voluntary way was the cause of more serious injuries in adults.[11] The ingestion of caustic products continued to be a serious public health problem that implied important functional sequelae and suicide attempts with caustics generate greater alterations at the level of the esophagus and stomach, for this reason, it was important to know the type and amount of substance ingested, the time elapsed from its consumption until the moment of receiving medical attention, if the ingestion was accidental or intentional and if the patient had been administered any product to neutralize or dilute matter or digested liquid.[9,12]

The main purpose of emergency management was patient survival and then all efforts should focus on treatment of early complications, prevention of delayed sequelae, preservation of nutritional autonomy and quality of life.[13] The therapeutic approach to caustic/acid injuries had evolved according to the understanding of cellular processes, therefore emetic agents and gastric lavage were avoided, which would cause re-exposure to the causative agent, so initially the pathway must be maintained. Permeable airway, monitor and replace possible volume depletion as well as potential septic conditions.[14] Sometimes a tracheostomy must be performed when the laryngeal damage was of great magnitude, a series of pediatric cases reported that 12% required intubation.[15] Other considerations for management included basic life support measures, analgesics, mucosal protectors (sucralfate), mitomycin C, proton pump inhibitors and anti-H2, studies such as tomography and endoscopy should be performed in the first 6-12 hours of the intake.[12,16]

According to the WSES guidelines emergency (2019) endoscopy should be performed if CT was unavailable, CT with contrast administration was contraindicated, CT suggested transmural esophageal necrosis but interpretation was difficult/uncertain or in the pediatric population (grade 2A).

Endoscopy used to be the mainstay of management algorithms following caustic ingestion, but the major drawback of endoscopy was its inability to predict accurately transmural necrosis, which may expose patients to either futile surgery or inappropriate watch and wait management and risk of death.[17]

Endoscopic balloon dilatation was the upfront treatment of esophageal and pyloric strictures, corrosive strictures can involve all esophageal segments and endoscopic dilation was the first-line management option.[18,19] There was sparse data on the endoscopic management of GOO/pyloric stenosis. Caustic-induced GOO can be managed using EBD with 97.3% clinical success. EBD can successfully ameliorate symptoms in patients with caustic-induced GOO, thereby avoiding surgery in most of the patients. EBD should therefore be the mainstay of management in such patients.[18]

CT scan was superior to traditional endoscopy for stratification of patients to emergency resection or observation.[13] In the event of massive bleeding, perforation or peritonitis, excision of the injury site should be considered including a jejunostomy to ensure enteral feeding. Surgical treatment was reserved for the chronic phase of ingestion, when esophageal (pharyngolaryngeal) strictures, gastroduodenal strictures or both develop and endoscopic dilations have failed. Surgical possibilities included transposition of the colon and the use of a segment of ileum, with surveillance in the intensive care unit.[9]

Endoscopy played a crucial role in the diagnosis, assessment of severity, treatment and follow up in patients with caustic injuries that may be difficult to dilate due to their complex anatomy and extensive fibrosis. Both CT and endoscopy were reliable diagnostic tools and their use should be tailored to the patient's condition. The advent of interventional endoscopy had renewed the interest of intraluminal stenting, but solid data supporting this approach was still lacking.[17]

CONCLUSION

The ingestion of caustic/acid substances represents a public health problem, the worldwide prevalence in children is accidental ingestion while intentional ingestions for self-harm purposes predominate in adults. It requires multidisciplinary management, where the treatment of sequelae is based mainly on balloon dilation, stent placement and/or complex reconstructive procedures. In this case, as in most patients with caustic-induced GOO can be successfully treated by EBD. Public health programs should be considered and easy access to strong corrosive agents should be limited.

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