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ORIGINAL ARTICLE

Gastric perforation after corrosive ingestion

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Abstract
Purpose To describe a series of children with gastric perforation (GP) after corrosive ingestion.
Methods Case notes of children treated for GP complicating corrosive ingestion between May 2001 and April 2010 were retrospectively reviewed.
Results Seventy-six children with corrosive ingestion were treated during the study period of which 5 (6.6%) developed GP. This complication was evident on admission in one case and developed within 48 h in the others. The major clinical findings were abdominal pain, tenderness, and distension with radiologic evidence of pneumoperitoneum. Associated pathology included necrosis of the abdominal esophagus in one case and duodenal perforation in another. Two cases have died during surgery while three survived with free of complications related to GP repair. Two patients developed gastric outlet obstruction (one with an esophageal stricture) on follow-up.
Conclusions GP is a rare but major complication of corrosive ingestion. Children who swallow corrosives should be closely monitored and pediatric surgeons should be aware of this potential early complication. The possibility of associated pathology should be considered when undertaking surgical repair.

Keywords Corrosive ingestion · Gastric perforation · Children

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Introduction

Accidental ingestion of corrosive substances, which may be life-threatening, is a continuing major health challenge for children. The esophagus is the most commonly affected site of the gastrointestinal tract after corrosive ingestion [1]. Development of clinically significant gastric injuries is relatively uncommon in children. Gastric outlet obstruction (GOO) is the most frequently reported gastric complication after corrosive ingestion in pediatric patients [2]. Gastric perforation (GP) is a very rare but significant and sometimes mortal consequence of corrosive ingestion [3–6]. GP following corrosive ingestion is not uncommon in adults [3–6]. However, to the best of our knowledge, there are only four pediatric case reports in the recent literature, and information about the presentation and management of this complication is therefore limited [7–9]. Pediatric surgeons need to be aware of this rare but serious possibility in children who swallow corrosive agents. We report five children with GP complicating accidental corrosive ingestion.

Patients and methods

Case notes of children who were treated for GP due to corrosive ingestion in our department between May 2001 and April 2010 were reviewed retrospectively. The following information was collected: demographic details, type of ingested agent, interval between ingestion and admission, interval between admission and diagnosis of GP, signs and symptoms, radiographic findings, leucocyte count, medications, operative findings, associated injuries and complications, surgical management, and outcomes.

Results

Seventy-six cases were admitted to our department following corrosive ingestion during the study period. Five (6.6%) of these patients developed GP. The type of ingested agent was liquid acid (lime remover) with unknown concentration in each GP cases. Initial treatment for corrosive-ingested children consisted of permitting nothing by mouth, intravenous fluids and broad spectrum antibiotics. A laryngoscopy prior to endotracheal intubation did not reveal any laryngeal injuries in our cases. An esophagoscopy was performed prior to the laparotomy in four children. The patients' data are summarized in Table 1. Figure 1 shows perforated stomach in Case 3. Follow-up ranged from 4 months to 4 years.

Discussion

Lime removing agents, which are strong acids, are widely used for household cleaning in our country. These liquids are mostly sold in safe childproof containers. However, they are also freely available on the shelf in grocery stores, especially in suburban districts. Acid concentration of these lime removers are not known. Their consumers buy these chemicals and often store them in standard drinks bottles in home kitchens. The colorless appearance of these liquids and their storage in used drinks bottles encourages their accidental ingestion by children.

There are conspicuous differences in the mechanism of injury, the maximum damage, and clinical presentation between ingested acids and alkalies. Transit time from mouth to stomach for swallowed acidic liquids is shorter than alkalies. Thus, while alkalies cause more severe injuries in oropharynx and proximal esophagus, acids are tend to develop more significant injuries in the distal esophagus and stomach [2, 7–9]. However, these differences in the injury mechanisms are not strict rules. Gastric injuries, as well as GP, may also develop due to alkali ingestions [3, 7, 10]. Severity of gastric injury is determined by several factors including amount and concentration of the ingested corrosive and fullness of the stomach at the time of ingestion [2, 6, 9]. The acid concentrations of the ingested agents were not known in our cases. We had information about the proximate amount of the ingested corrosives in three cases, in whom the volume of the ingested acid was between 10 and 30 ml. In our study, a delayed GP developed 36–52 h later in the children who had a meal 1–2 h prior to corrosive ingestion. However, GP was evident on admission in Case 3, who had a meal 4 h prior to corrosive ingestion. The relationship between fullness of the stomach at the time of acid ingestion and time of development of gastric injury in presented series was consistent with the general knowledge.

The interval between corrosive ingestion and development of a delayed GP may vary both in children and adults [6, 9, 11]. Gillis et al. [9] reported a 2-year-old boy in whom a delayed GP developed 7 days after soldering ingestion. In a 29-year-old case of hydrochloric acid ingestion, GP developed 2 days later [11]. Zargar et al. [6] reported that one of their five adult GP cases developed perforation 8 days after acid ingestion. In our study, perforation was evident on admission in one child. GP delayed 20–52 h in our four patients. Thus, we conclude that pediatric surgeons should be aware of the possibility of a delayed GP after corrosive ingestion. High index of suspicion and close monitoring of these patients are essential points for diagnosis of a delayed GP in a timely manner.

As in the presented cases in this study, the children with GP due to corrosive ingestion show classic clinical and radiologic signs and findings of gastrointestinal perforation such as abdominal pain, tenderness, and distension [4, 7–9]. The patients frequently show life-threatening symptoms such as hypotension, dyspnea, and severe edema in oropharyngeal mucosa [7, 8]. In serious cases, respiratory arrest may develop [8]. Blood-tinged vomitus, hemoptysis, and skin burns may be present [3, 4, 8]. In our series, the patients almost always showed severe symptoms or additional injuries. Three cases had significant respiratory failure requiring ventilatory support. One patient had second- and third-degree skin burns. Hematemesis was an alerting symptom in one case. Presence of such serious signs and associated injuries in children with corrosive ingestion should alert the pediatric surgeon for the possibility of GP. In their adult series, Jeng et al. [12] concluded leucocytosis as a risk factor for a serious injury. However, we encountered normal leucocyte count in one patient who has died. White blood cell count was significantly high only in one of our cases. Plain abdominal X-ray is an useful radiologic tool in corrosive ingested patients to confirm a GP by detecting pneumoperitoneum [7–9]. However, as in our cases, an initial abdominal X-ray on admission may be negative for free intraperitoneal air, and a repeated radiogram may be helpful for diagnosis of GP. Kanne et al. [11] suggested that computerized tomography can help in the immediate and subsequent evaluation of gastric injury and perforation following caustic ingestion. Initial flexible endoscopy may demonstrate a gastric injury at an early stage [3, 5].

It was claimed that the antrum and pylorus are more frequently affected than corpus and fundus of the stomach [6, 7]. We have observed a pyloric perforation and a pyloric plus antral perforation in one case, respectively. However, perforation was located in the fundus and corpus of the stomach in the remaining three patients. The lesion

Table 1 Characteristics of the patients

Case	Age sex	T1 (h)	T2 (h)	Quantity of CA	Hours after meal	Symptoms suggestive for GP	Abdominal X-ray at admission	Resp. Dist.	MA	WBC ($\times 10^3$)	Gastric pathology	Additional injury	Long-term complication	Management	Outcome
1	1.5 F	4	36[a]	Not known	Not known	Rapid abdominal distension, pneumoperitoneum on X-ray	No	Yes	Yes	6.3	Necrosis and perforation of fundus and corpus	Grade 2 esophageal burn, partial necrosis of abdominal esophagus		She has died during laparotomy for primary repair	Exitus
2	1.5 M	14	12[a]	Not known	2 h	Development of abdominal pain and tenderness during observation, pneumoperitoneum on X-ray	Yes (Normal)	Yes	Yes	25.8	Necrosis and perforation of antrum and pylorus	Perforation of proximal duodenum		He has died during laparotomy for primary repair	Exitus
3	11 M	1	1	15–20 ml	4 h	Abdominal pain and tenderness, mild abdominal distension, pneumoperitoneum on X-ray	Yes (SDA)	No	No	11.3	A 6 × 5 cm perforation in the fundus and corpus	Grade 2 esophageal burn	GOO at third week, esophageal stricture at fifth week	Primary repair (G/J for GOO and repeated dilatations for esophageal stricture)	No complication for GP. Lost to follow-up after 1 year
4	1.5 F	4	48	20–30 ml	1 h	Rapid abdominal distension during observation, pneumoperitoneum on X-ray	Yes (Normal)	Yes	No	11.0	A 6 × 6 cm perforation in the fundus and corpus	Skin burns on the neck and arm. Grade 2 esophageal burn	GOO at fourth week	Primary repair (Heineke–Mikulicz pyloroplasty for GOO)	Doing well
5	5 M	1	36	10–15 ml	2 h	Hematemesis, abdominal pain, tenderness and rapid distension during observation, pneumoperitoneum on X-ray	Yes (Normal)	No	No	13.8	A 3 × 2 cm perforation in the antrum	Grade 2 esophageal burn		Primary repair	Doing well

F female, *M* male, *T1* duration between ingestion and admission, *T2* duration between admission and diagnosis, *CA* corrosive agent, *GP* gastric perforation, *SDA* subdiaphragmatic free air, *Resp. Dist* respiratory distress, *WBC* white blood cell count, *MA* metabolic acidosis, *GOO* gastric outlet obstruction, *G/J* gastrojejunostomy

[a] Diagnosed to have gastric perforation while under observation for a presumptive diagnosis of acid poisoning at the pediatric intensive care unit

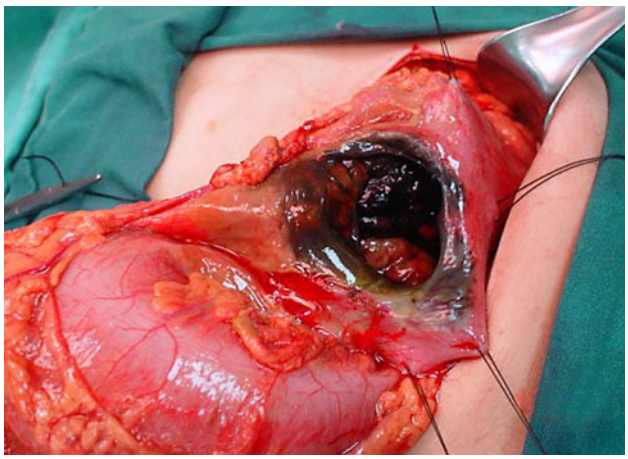

Fig. 1 Operative photograph of Case 3 showing the site of gastric perforation

was located on the anterior side of stomach in two cases, while both sides of the organ were affected in three patients.

Additional gastrointestinal tract injuries such as esophageal perforation, duodenal and jejunal necrosis and perforation may present with GP [5–7, 10, 12]. We encountered esophageal injuries in four children. Three patients were diagnosed to have grade 2 esophageal burn that was confirmed by esophagoscopy. Laparotomy revealed complete necrosis of esophagus at the esophagogastric junction in one case. Necrosis and perforation of the first part of duodenum was another significant associated gastrointestinal injury among our patients. A detailed preoperative evaluation of the patient and careful exploration of abdominal organs during laparotomy may prevent the child from overlook of the additional injuries.

Depending on the type of injury, different surgical techniques, such as primary repair of the GP and total gastrectomy with esophagojejunostomy were performed for treatment of GP cases [7, 8]. A laparotomy was performed in all our cases. Debridement of necrotic tissues and primary repair of stomach was performed. We were unable to complete surgery due to intraoperative cardiac arrest in two cases. Gastric narrowing, stricture of anastomosis are possible long-term complications of surgical repair [6, 7]. In this study, the surviving patients did not develop any early or long-term complication that can be attributable to GP repair. However, GOO and esophageal stricture were two distinct long-term morbidities related to acid ingestion in our cases. GOO is a well-known late gastric complication of corrosive ingestion, and develops due to pyloric scarring formation [2]. We have observed GOO in two patients. In both of these children, GP was in the fundus and corpus of the stomach, and pylorus was found macroscopically normal at operation. Therefore, we believe that GOO was not a complication of primary gastric repair. A gastrojejunostomy and a pyloroplasty have been done in each GOO case, respectively.

Mortality rate in patients with corrosive induced GP is considerable [3, 5, 6]. Zargar et al. [6] reported three deaths among four GP cases. Berthet et al. [3] noted 3 deaths because of extensive necrosis of stomach in their 10 patients, who were surgically repaired by esophagogastrectomy. High mortality rate in the adults was attributed to intentional ingestion of high amount of corrosives for suicide [5, 6]. Jeng et al. [12] suggested that the main causes of operative mortality are multiple organ failure and persistent metabolic acidosis. Even mortality was not reported in four pediatric cases [7–9], and two out of our five cases with GP have died. We attributed these two deaths to chemical peritonitis, metabolic acidosis, and respiratory failure.

In conclusion, GP following corrosive ingestion is a rare but significant complication in childhood. Even ingestion of acid substances more frequently lead to GP and children with alkali substance ingestion are under risk, as well. Pediatric cases with severe symptoms following corrosive ingestion should be closely monitored for a possible GP, which may develop in an early or a delayed manner. Delayed diagnosis, presence of respiratory distress, and metabolic acidosis maybe major prediction points for a worse outcome. Debridement of necrotic tissues and primary repair of stomach is safe in selected patients, however, more aggressive procedures such as total gastrectomy and esophagojejunostomy may be required. Close follow-up of the patients after discharge from the hospital is mandatory for detection of possible long-term complications such as GOO and esophageal strictures. Education of parents is an important tool for prevention of children from corrosive injuries. New legislations for restriction of easy access to non-standard corrosive household cleaning agents by prohibiting their free commercialization are required.

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