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Gastric outlet obstruction due to corrosive ingestion: incidence and outcome

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Abstract A retrospective clinical study was performed to determine the incidence, management, and outcome of gastric outlet obstruction (GOO) caused by caustic ingestion in children. Of 220 patients who sustained caustic substance ingestion and were treated at our unit between 1976 and 1996, 168 ingested alkaline substances; of these, 9 children (5.3%) developed GOO in addition to esophageal strictures. The remaining 52 patients ingested acid agents, and 2 of them (3.8%) presented with GOO without esophageal strictures. The overall incidence of corrosive GOO was 5% (n = 11). The mean age of the patients with GOO was 5.7 ± 2.8 years (range 2–14) with a female:male ratio of 6:5. Sodium hydroxide (n = 6), potassium hydroxide (n = 3), and hydrochloric acid (n = 2) were the ingested caustic agents. The patients were subdivided into two groups according to serial endoscopic and radiologic findings: group I: moderate (dense superficial and spotty ulcerations with intact mucosa) mucosal injury with partial pyloric obstruction; and group II: severe (deep ulcerations, extreme hemorrhagic erosions, eschar formation with white plaques) mucosal injury with complete pyloric obstruction. Group I consisted of 5 patients who ingested alkali agents while group II included 6 who presented with ingestion of alkaline (n = 4) and acid (n = 2) agents. Surgical treatment included Billroth I (n = 6) operations performed in group II and Finney (n = 3) and Heineke-Mikulicz (n = 2) pyloroplasty procedures done in group I. All patients are alive without any complaints. Fiberoptic endoscopy should be the preferred method of evaluating a patient with ingestion of a corrosive agent. It determines the presence of injury and assesses the extent of damage, establishing the diagnosis and allowing therapy to be instituted immediately. Our experience revealed that substantial damage has occurred early after ingestion, and early surgical intervention has decreased the morbidity and mortality. The extent of the mucosal injury and status of the pylorus and antrum determined the type of surgical treatment. A Billroth I procedure recommended for severely injured mucosa with complete pyloric obstruction, and pyloroplasty for moderate mucosal injury associated with partially obstructed but still viable pylorus. In contrast to the current belief, alkali ingestion also has a high risk of corrosive gastric injury causing GOO, which should be considered during assessment of the injury. We emphasize that a detailed evaluation of radiologic and especially endoscopic findings is very important for determining the timing, necessity, and type of appropriate surgical treatment.

Key words Gastric outlet obstruction · Corrosive ingestion · Alkali ingestion

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Introduction

Accidental caustic substance ingestion is one of the common worldwide problems among children. The most important complications are esophageal and gastric burns, which may result in severe strictures [13]. In spite of the different treatment methods described for prevention of stricture formation, a considerable percentage of patients sustaining caustic ingestion develops esophageal and/or gastric strictures [7]. Although detailed information is available about corrosive esophageal strictures [8] and various other causes of gastric outlet obstruction (GOO) [6], very little is known about the incidence, clinical course, and outcome of GOO due to corrosive ingestion in children. Therefore, a retrospective clinical study was performed to determine the incidence, management, and outcome of GOO caused by caustic ingestion in children.

Patients and methods

The records of all patients treated for ingestion of caustic substances during a 21-year period between 1976 and 1996 inclusive at

the Department of Pediatric Surgery of Hacettepe University Medical Faculty were reviewed with regard to the development of GOO. Information recorded for each patient included age, sex, type of ingested agent, diagnostic studies, treatment methods, and outcome.

Results

Of 220 patients who sustained caustic substance ingestion and were treated at out unit, 162 were male (73.6%) and 58 were female (26.3%); 188 (85.4%) were younger than 6 years of age at presentation. One hundred sixty-eight children ingested alkaline substances, and 9 of these (5.3%) developed GOO in addition to esophageal strictures. The remaining 52 ingested acid agents, and 2 of them (3.8%) presented with GOO without esophageal strictures. The overall incidence of corrosive GOO was 5% (n = 11). The mean age of the patients who presented with GOO was 5.7 ± 2.8 years (range 2–14) with a female:male ratio of 6:5. Accidental ingestion was noted in 9 cases, while 2 were suicide attempts. Sodium hydroxide (n = 6), potassium hydroxide (n = 3), and hydrochloric acid (n = 2) were the ingested agents.

The diagnosis was made by upper gastrointestinal (GI) contrast series and esophagogastroscopy, which were done within 1 to 8 days after caustic ingestion in all patients. Medical treatment consisting of steroids, antibiotics, and histamine-2 (H_2) antagonists was commenced after the diagnosis was made. The patients were subdivided into two groups according to the pertinent serial endoscopic (13) and radiologic findings as: group I: moderate (dense superficial and spotty ulcerations with intact mucosa) mucosal injury with partial pyloric obstruction (Fig. 1a); and group II: severe (deep ulcerations, extreme hemorrhagic erosions, eschar formation with white plaques) mucosal injury with complete pyloric obstruction (Fig. 2a).

Group I consisted of 5 patients who ingested alkaline agents while group II included 6 who presented after ingestion of alkaline (n = 4) and acid (n = 2) agents. The interval for the development of GOO ranged from 16 to 35 days (mean 27 days) in group I and 7 to 15 days (mean 11 days) in group II. Upper GI series revealed the stomach to be distorted, minimally distensible, and lacking peristalsis to variable degrees in all patients. Nausea, vomiting, bloating, postprandial epigastric fullness, and pain were the common signs and symptoms in all cases. The serum electrolyte and acid-base balance alterations were those of extracellular volume depletion and metabolic, hypochloremic alkalosis.

Surgical treatment included Billroth I (n = 6) operations performed in group II and Finney (n = 3) and Heineke-Mikulicz (n = 2) pyloroplasty procedures in group I. Billroth-type operations were performed at an average of 1.5 months following presentation while the interval was from 2 to 4 months for pyloroplasty procedures. Histopathologic evaluation of specimens revealed islands of gastric mucosal epithelium with hemorrhage and inflammation of the lamina propria, severe edema of the submucosa, and dense fibrosis of the muscular layers.

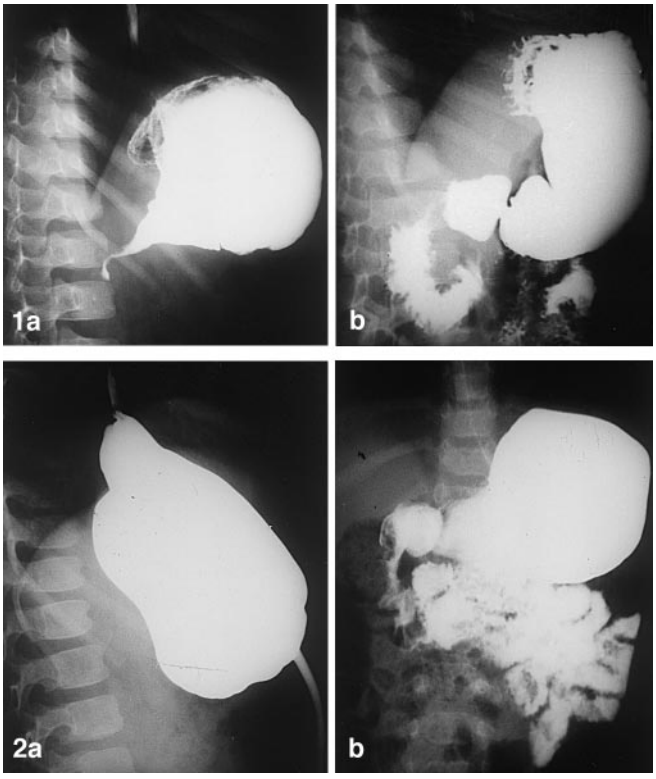

Fig. 1 **a** Preoperative upper gastrointestinal (UGI) series revealing partial pyloric obstruction. **b** Postoperative UGI series of same patients showing passage of contrast through gastroduodenal junction

Fig. 2 **a** Preoperative upper gastrointestinal (UGI) series revealing complete pyloric obstruction. **b** Postoperative UGI series of same patient showing passage of contrast through gastroduodenal junction

The efficacy of the surgical treatment was evaluated by clinical assessment associated with endoscopic and radiologic studies (Figs. 1b and 2b) every 6 months in the first 2 years. The follow-up period ranged from 3 to 15 years with a mean of 9 years. Retrograde and/or antegrade dilation for esophageal strictures was successful in 6 patients, while 3 children who ingested alkalis required right colon interposition for esophageal replacement. All patients are alive without any complaints.

Discussion

Corrosive GOO has been considered an uncommon entity with an estimated concomitant incidence of 20% of all corrosive esophageal strictures [9]. It is known that ingestion of alkali results primarily in oropharyngeal and esophageal damage, while acids tend to spare the esophagus and produce coagulation necrosis of the stomach as in a thermal burn. The esophagus is spared because of the relative resistance of squamous epitheli-

um to acid damage and the short duration of contact due to the rapid transit of acid through the esophagus [7, 8, 13]. The acid pools in the gastric antrum or other dependent portions of the stomach, and may produce severe mucosal damage [9, 10]. However, our findings revealed that alkali ingestion also carries a high risk of severe gastric damage. The factors determining the extent of corrosive gastric injury are the nature of the agent, amount and concentration of the ingested material, mode of ingestion, position of the patient during ingestion, and whether it was taken in the fasting or fed state [5, 10]. Theses factors result in a wide clinical variability of corrosive gastric injuries associated with esophageal stricture, and cause controversies in diagnostic and treatment approaches [3, 12].

The risk of instrumental perforation of the esophagus and/or stomach following ingestion has been the principal deterrent to routine early endoscopy [1]. However, with the introduction of fiberoptic endoscopes, the risk of perforation is theoretically much reduced. According to our experience, fiberoptic endoscopy should be the preferred method of evaluating a patient with ingestion of a corrosive agent. Since severe changes may occur with minimal symptoms, physical findings, or radiologic changes, endoscopic evaluation determines the presence of injury and assesses the extent of damage, thereby establishing the diagnosis and allowing therapy to be instituted immediately. If severe esophageal lesions of doubtful extent are encountered during endoscopy, the examination must be terminated and then repeated at 48-h intervals to examine the stomach and duodenum at early stage.

Endoscopy is also helpful in determining the degree and character of the damage for optimum timing of surgical intervention. Endoscopy should be credited as a life-saving procedure in the presence of black discoloration indicating mucosal gangrene with full-thickness necrosis. Prompt surgical resection with primary or delayed reconstruction obviates the risk of sepsis arising from the necrotic foci or a neglected perforation, the single most frequent cause of mortality in most reported series [5]. Radiologic studies including upper GI series are also essential for detecting perforations late sequelae, and the motility of the esophagus, stomach, and duodenum.

The most controversial aspect of management deals with the timing, necessity, and type of surgical intervention. The timing and necessity of surgery is mainly determined by the nature of the symptoms and degree of GOO as demonstrated radiologically and by gastroscopy. In order to prevent inadequate resection of the stomach, some authors have advised against early surgery [2, 4]. However, our experience revealed that substantial damage has occurred early after ingestion, and early surgical intervention has decreased the morbidity and mortality. Additionally, delaying operation after acid ingestion may cause the damage to extend within the stomach or beyond, since concentrated acid is not readily neutralized [3].

A detailed evaluation of the endoscopic and radiologic findings is the most helpful tool for the choice of the optimum surgical method. The extent of the mucosal injury and status of the pylorus and antrum determined the type of surgical treatment in our series. A Billroth I procedure is recommended for severely injured mucosa with complete pyloric obstruction and pyloroplasty for moderate mucosal injury associated with a partially obstructed but still viable pylorus. Additionally, a gastrostomy should be performed in patients presenting with severe esophageal injuries so that safer retrograde esophageal dilations and enteral nutrition can be instituted.

Although pyloroplasty procedures are thought not to be adequate long-term solutions since progressive fibrosis may occur, we have not encountered any problems requiring surgical treatment during long-term follow-up. Thus, we recommend pyloroplasty procedures for moderate forms of corrosive gastric injury causing partial GOO. Resection of the nonviable gastric tissue appears to provide the most favorable long-term result in patients with severe gastric injury presenting with complete pyloric obstruction. Gastroenterostomy procedures without resection of the injured, nonviable gastric tissue are not recommended due to the risk of development of carcinoma, metaplasia, and late marginal ulceration.

The intervals for development of GOO in our series were within the limits stated in the literature, which vary from 7 days to 3 years [4, 9, 11, 12]. Late sequelae of corrosive gastric injury such as achlorhydria, protein-losing gastroenteropathy, duodenal atonicity, mucosal metaplasia, and gastric carcinoma have not been encountered in our series, although they have been reported in adult series [11]. We are not sure whether the absence of late sequelae is because of the relatively shorter long-term follow-up or the ingestion of small volumes as a characteristic of children. Additional data are required to properly evaluate this finding.

Our experience has revealed that, in contrast to the current belief, alkali ingestion also has a high risk of corrosive gastric injury causing GOO, which should be considered during assessment of the injury by endoscopic and radiologic studies. We emphasize that detailed evaluation of the radiologic and especially the endoscopic findings is very important for determining the timing, necessity, and type of appropriate surgical treatment.

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