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Yasar Dogan, Tülay Erkan, Fügên Çullu Çukurgas and Tufan Kutlu

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Caustic Gastroesophageal Lesions in Childhood: An Analysis of 473 Cases

Yaşar Doğan, MD
Tülay Erkan, MD
Füügen Çullu Çokuğraş, MD
Tufan Kutlu, MD

Summary: Ingested corrosive agents produce oropharyngeal and gastroesophageal injuries ranging from minor burns to severe necrosis, depending on the agent amount, concentration, and duration of exposure. The aim of this study was to present our patients with corrosive ingestion retrospectively. Four hundred seventy-three children younger than 16 years of age (mean age, 3.7±0.1 years) who were admitted to our hospital for suspected corrosive ingestion between the years 1995 and 2003 were studied. Two hundred eighty-six (60.5%) of 473 patients were males. Household bleaches (36.6%) and oven cleaners (23%) were the most frequently encountered corrosive agents. During endoscopy, lesions in the esophagus were recorded in 379 children. Eighty-one of the cases had gastric lesions. During the follow-up, esophageal stricture, esophageal perforation, and gastric outlet obstruction (GOO) developed in 11 cases, 1 case, and 2 cases, respectively. Caustic ingestion of alkali substances such as oven cleaner seem to cause more severe injuries. Early admission to the hospital with clinical and endoscopic evaluation and early surgery when required may reduce morbidity and mortality. Clin Pediatr. 2006;45:435-438

Introduction

Accidental ingestion of caustic agents, potentially capable of burning the esophagus, is generally a problem of childhood. Young toddlers especially, who are investigating their surroundings and are unaware of many dangers, are prone to this kind of accident.1,2 The clinical manifestations of caustic ingestion in children vary from no injury to fatal outcome.3,4 One of the important complications of caustic ingestion is corrosive esophagitis, which may lead to stricture formation.5 The outcome of corrosive injuries may be associated with caustic properties, extent of esophageal burns, and treatment modalities.6 The aim of this study was to evaluate the records of all children with suspected caustic ingestion admitted to our hospital between the years 1995 and 2003.

Materials and Methods

Four hundred seventy-three children younger than 16 years of age (mean age, 3.7±0.1 years) who were admitted to our hospital for suspected corrosive ingestion were enrolled in this study. The parameters examined included the year of admission, age, sex, caustic agent, degree, and site of burn. All patients underwent endoscopic examination...
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within the first 48 hours. The grading of esophagitis was classified based on the endoscopic findings as “no injury,” “grade I burns” (superficial erythema or mucosal hyperemia), “grade IIa and grade IIb burns” (shallow linear and circular ulcers limited to mucosa), “grade III burns” (ulceration deep into the muscle or transmural perforation). After treatment, control endoscopic examinations of all patients were performed within 10 days. All patients with grade IIb and grade III burns received methylprednisolone (2 mg/kg/day; maximum, 60 mg/day for 3 weeks), ranitidine, and ceftriaxone. Second and third control endoscopies were performed within the 3rd and 4th week, respectively. Early and late complications were noted.

Results

Of the 473 patients, 286 (60.5%) were males, 187 (39.5%) were females, and the ages of the patients ranged from 15 days to 16 years (mean age, 3.7±0.1 years). Household concentrated bleaches (n, 173; 36.6%) and oven cleaners (n, 109; 23%) were the most frequently encountered corrosive agents. Forty-eight children had drunk toilet bowl cleaners, 43 lime solvents, 30 hydrochloric acid, 18 potassium permanganate, and 17 acetic acid. The remaining 35 children had drunk a variety of agents (Table 1). In 43 of the patients, corrosive agents had been given by their mothers accidentally. The remaining 427 patients had drunk corrosives accidentally and 3 for suicide. After ingestion of the corrosive agents, 208 patients had vomited. During endoscopy, lesions in the esophagus were seen in 379 children. In 225 of these patients, only slight changes (grade I and IIa) were present; whereas in 154 patients, the lesions were more severe (grade IIb and III). Eighty-one of the patients had gastric lesions. Most of the patients with gastric lesions had drunk oven cleaners, lime cleaners, and concentrated bleach (Table 1). Although oral cavity burns were not seen in 389 patients, endoscopy revealed esophageal lesions in 240 of them (61%). Of the 379 children with esophageal burns, only 154 with grade IIb and grade III burns received antibiotics, steroids, and H₂ receptor blockers. After the second and third endoscopy, esophageal stricture was observed

| Table 1CORROSIVE AGENTS AND THE FIRST ENDOSCOPY FINDINGS |
|----------------------------------|------------|------|-------|-------|-------|-------|-------|-------|
| Corrosive Agents | No | % | N | GI | GIIa | GIIb | GIII | GL |
| Concentrated bleach | 173 | 36.6 | 52 | 83 | 22 | 16 | 0 | 11 |
| Oven cleaner | 109 | 23 | 11 | 15 | 15 | 58 | 10 | 24 |
| Toilet bowl cleaner | 48 | 10.1 | 8 | 14 | 11 | 12 | 2 | 9 |
| Lime solvent | 43 | 9.1 | 7 | 12 | 8 | 14 | 3 | 14 |
| Drain cleaner (HCL) | 30 | 6.3 | 3 | 6 | 5 | 13 | 3 | 7 |
| Potassium permanganate | 18 | 3.8 | 4 | 8 | 2 | 2 | 2 | 0 |
| Acetic acid | 17 | 3.6 | 4 | 4 | 1 | 8 | 0 | 6 |
| Benzalkonium chloride | 12 | 2.5 | 0 | 2 | 6 | 3 | 1 | 0 |
| Hydrogen peroxide | 7 | 1.5 | 1 | 1 | 2 | 2 | 1 | 5 |
| Hair dyes | 5 | 1.1 | 0 | 0 | 3 | 2 | 0 | 1 |
| Antifreeze | 2 | 0.4 | 0 | 0 | 0 | 1 | 1 | 2 |
| Other ingestion substances | 9 | 1.8 | 4 | 3 | 2 | 0 | 0 | 2 |
| Total | 473 | 100 | 94 | 148 | 77 | 131 | 23 | 81 |

N: normal; GI: grade I; GIIa: grade IIa; GIIb: grade IIb; GIII: grade III; GL: gastric lesions.
in 11 patients, esophageal perforation in 1 patient, and gastric outlet obstruction (GOO) in 2 patients. All patients with esophageal stricture had drunk oven cleaners. Esophageal perforation was observed in 1 patient who had ingested hydrochloric acid. Gastric outlet obstruction developed in 2 patients who had ingested hydrochloric acid and oven cleaners. Seven patients with esophageal stricture could be dilated by balloon without complications. The remaining 4 patients developed esophageal perforation during dilatation procedures. Two of them were treated medically and further dilatations were carried out. The third patient underwent resection of the stenotic part of the esophagus, and the last patient with gastrostomy was scheduled for interposition surgery. Pyloroplasty was performed in two other patients with GOO.

Discussion

Corrosive injury caused by ingestion of caustic substances is a worldwide pediatric emergency problem. More than 100,000 children suffer corrosive esophageal burns every year in the United States. The incidence of corrosive ingestion in Turkey is still unknown.

The highest risk group for accidental caustic ingestion is children younger than 5 years old. In this study, the mean age was 3.7 years, and the male-to-female ratio was approximately 1.5. The extent and severity of the caustic injury to the esophagus depends on the interactions of four factors: the corrosiveness of the ingested substance, its quantity and concentration, the duration of contact time, and subsequent secondary infection. Because alkaline agents produce liquefaction necrosis and rapid penetration, they often cause more severe injuries than acidic agents in the esophagus. In this report, the most accidents involving caustic ingestion were caused by alkalines (e.g., oven cleaner), as in the other studies. Early esophagogastroscopy performed within 24 to 36 hours after the injury is extremely important for assessing both the degree of esophageal burn and the protocol for subsequent treatment. All patients underwent endoscopic examination within the first 48 hours. Esophageal lesions were recorded in 80.1% and gastric lesions in 17.1% of all cases. Wijburg et al in their study had found esophageal lesions in 39% of the children who were suspected of having ingested a caustic agent. Several studies indicated that the clinical manifestations of caustic ingestion injuries are poor predictors of the extent or depth of esophageal injury. Crain et al found that only 33% of those patients with evidence of oral cavity burns actually had esophageal burns. In our study, esophageal lesions were seen in 61% of the 389 patients who did not have any oral cavity burns. Medical management of caustic ingestion includes antibiotics, steroids, and H2 receptor blockers. Antibiotics have been shown to markedly reduce the incidence of stricture formation. The use of steroids in caustic esophagitis is controversial. Most authors agree that patients with first- or third-degree esophageal injuries do not benefit from steroid use. The effect of steroids in preventing stricture formation in second-degree burns is debatable. In this study, all cases were treated with steroids (3 weeks), ranitidine, and antibiotic. The incidence of esophageal stricture as a consequence of caustic ingestion varies at 2% to 63% in different studies. Complications developed in 14 (2.9%) of our patients; alkaline ingestion was responsible for 85.7% of complications, as in the other studies. The traditional primary treatment for caustic esophageal strictures is dilatation. If conservative management with esophageal dilatation failed to provide adequate relief from symptoms, surgical esophageal replacement by retrosternal right colonic interposition was performed. Seven of 11 patients with esophagus stricture could be dilated by balloon without complications. The remaining 4 patients developed esophageal perforation during the dilatation procedure. Two of them were treated medically and further dilatations were carried out. The third patient underwent resection of the stenotic part of the esophagus, and the fourth patient with gastrostomy was scheduled for interposition surgery. In 2 patients with GOO, pyloroplasty was performed. In conclusion, caustic ingestion of alkali substances such as oven cleaner seems to cause more severe injuries.

REFERENCES


