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Gastrointestinal system lesions in children due to the ingestion of alkali and acid corrosive substances

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Background/aim: To examine esophageal and gastric lesions in children due to the ingestion of alkali and acid corrosive substances and to emphasize all related complications.

Materials and methods: The reports of 103 children who ingested or were suspected to have ingested corrosive substances and who then underwent upper gastrointestinal endoscopic inspections were evaluated retrospectively.

Results: Of the patients, the mean age was 41 ± 3.6 months, and 57.3% were male. Vomiting was the most common symptom (44.7%). Eighteen different commercial products were defined as corrosive substances: 59.2% of them were alkali, 39.8% were acids, and 1% had a neutral pH. These corrosive agents most frequently contained sodium hydroxide, followed by hydrochloric acid, sodium hypochlorite, and sulfuric acid. Endoscopic inspections were abnormal in 68% of the cases. Esophageal lesions were observed in 56.3% of the patients, while gastric lesions were observed in 35%. During the follow-up period, esophageal strictures developed in 4.9% of patients, while gastric outlet obstructions developed in 1%.

Conclusion: Of the patients, the mean age was 41 ± 3.6 months, and 57.3% were male. Vomiting was the most common symptom (44.7%). Eighteen different commercial products were defined as corrosive substances: 59.2% of them were alkali, 39.8% were acids, and 1% had a neutral pH. These corrosive agents most frequently contained sodium hydroxide, followed by hydrochloric acid, sodium hypochlorite, and sulfuric acid. Endoscopic inspections were abnormal in 68% of the cases. Esophageal lesions were observed in 56.3% of the patients, while gastric lesions were observed in 35%. During the follow-up period, esophageal strictures developed in 4.9% of patients, while gastric outlet obstructions developed in 1%.

Key words: Corrosive substance, endoscopic inspection, esophageal stricture, gastric outlet obstruction

1. Introduction

The ingestion of corrosive substances among children mostly occurs accidentally. Most of these corrosive agents are household cleaners, and the esophagus is the most damaged site of the gastrointestinal system. Perforation and death are potential serious complications in the acute period, while stricture and cancer can occur over chronic periods of time. Endoscopic inspection in the early hours after ingestion is very important for the evaluation of the lesions to allow the planning of the appropriate treatment and follow-up care of these patients (1–3).

In this study, we investigated children who were admitted to our clinic due to corrosive substance ingestion or suspected ingestion and who subsequently underwent an upper gastrointestinal endoscopy. The aim of this study was to examine esophageal and gastric lesions in children due to the ingestion of alkaline and acidic corrosive substances and to emphasize all relevant complications.

2. Materials and methods

One hundred and three children who were admitted to our pediatric gastroenterology unit during a 3-year period complaining of corrosive substance ingestion and who subsequently received upper gastrointestinal endoscopies were enrolled in this study. Clinical and endoscopic findings, treatment regimes, and long-term complications were retrospectively evaluated.

The endoscopic procedure was first performed with a 5-mm diameter flexible endoscopy device (Olympus Evis Lucera CLV-260SL) 12–24 h after the ingestion of the corrosive substance in the patients admitted to our clinic. Before the procedure, 0.1–0.4 mg/kg intravenous midazolam was administered to sedate the patients.

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All esophageal and gastric lesions were noted. The esophageal lesions were evaluated according to a classification of "corrosive esophagitis grading" (4). According to this classification, normal mucosa was accepted as Grade 0; mucosal hyperemia and edema as Grade I; superficial erosions, bleeding, and white membranes on the mucosa as Grade IIA; deep and circular ulcerations on the mucosa as Grade IIB; necrotic areas with multiple ulcers on the mucosa as Grade IIIA; widespread necrosis on the mucosa as Grade IIIB; and perforation as Grade IV. The gastric findings were noted according to the normal mucosal appearance, edema and hyperemia, superficial erosions, ulcer, and necrosis. The locations of the lesions according to the pH of the corrosive substance were compared using the Mann–Whitney U test. $P < 0.05$ was considered statistically significant.

3. Results

Of the patients, 59 (57.3%) were male and 44 (42.7%) female. Their mean age was 41 ± 3.6 months (range: 2–204 months). Corrosive substances were accidentally administered by siblings in the case of 2 patients and by the mother in the case of 1 patient; 2 patients had purposefully ingested corrosive substances due to suicidal intentions. Two patients had a mental disorder. The remaining 96 patients (93%) ingested corrosive substances by themselves accidentally.

The children complained of the following symptoms upon admission: vomiting, 46 (44.7%); cough, 5 (4.9%); blood in the mouth and/or vomiting with blood, 4 (3.9%); hypersalivation, 3 (2.9%); pain in the mouth, 3 (2.9%); wound in the mouth, 2 (1.9%); bitter taste in the mouth, 2 (1.9%); abdominal pain, 2 (1.9%); and difficulty swallowing, 1 (1%).

The corrosive substances were from 18 different commercial products and were alkaline in 61 cases (59.2%), acidic in 41 cases (39.8%), and of a neutral pH in 1 case. Sodium hydroxide was the most common corrosive substance (34%). Other frequent substances included the following chemicals: hydrochloric acid, sodium hypochlorite, and sulfuric acid. The observed endoscopic findings in the esophagus and stomach are presented according to the ingested corrosive substances in Table 1. The endoscopic examinations were determined to be normal in 33 (32%) cases and abnormal in 70 (68%). Of the cases with caustic lesions (n = 70, 68%), the lesions were found only in the esophagus in 34 (48.6%), only in the stomach in 12 (17.1%), and in both the esophagus and the stomach in 24 (34.3%).

Some photographs of the esophageal and gastric lesions of the cases are shown in Figures 1A–1E. The locations of the patients' lesions are compared in Table 2 according to the pH of the corrosive substances. The differences in the esophageal and gastric lesions according to the alkaline and acidic qualities of the corrosive substances were not statistically significance.

The patients were hospitalized for a mean duration of 3.4 ± 3.9 days (range: 1–30 days). Oral nutrition was stopped, and intravenous ranitidine and prophylactic antibiotherapy (ceftriaxone) were given to all patients. Intravenous pantoprazole was added to the treatment of the patients who had Grade IIB or worse esophagitis or gastric ulcers. Ceftriaxone and ranitidine treatment was ended for patients with completely normal endoscopic findings. The treatment for patients with detectable lesions, including Grade IIA or less severe esophagitis and mild gastric lesions, was continued with peroral ranitidine if the patients were under 1 year of age or with peroral lansoprazole if the patient was older than 1 year and had started oral nutrition. For the 27 (26.2%) patients with grade IIB or worse esophagitis and severe gastric lesions, oral nutrition was stopped and total parenteral nutrition was given for 5.1 ± 2.4 days (range: 3–12 days).

In the follow-up, esophageal strictures developed in 5 (4.9%) patients. Four of these patients had ingested sodium hydroxide and had Grade IIIA corrosive esophagitis upon their first endoscopic examinations. Recurrent bougie or balloon dilatation procedures were administered to these patients, and 1 of them had already undergone surgery at 2 years of age. The fifth patient, who developed esophageal strictures, had Grade IIB corrosive esophagitis upon his first endoscopic examination and had ingested drain cleaner. He underwent a balloon dilatation. A patient who ingested paint thinner was admitted to our clinic with a complaint of vomiting 30 days after the ingestion. Gastric outlet obstruction was observed in this case, and the patient was referred to surgery for pyloroplasty.

4. Discussion

The ingestion of corrosive industrial chemical agents, which are mostly used for household cleaning, usually occurs accidentally or sometimes for suicidal purposes. This issue represents an important health problem for the public, as people may not take sufficient preventive measures (2,3,5–7). It has been reported in the literature that 10%–15% of accidental ingestions happen due to inappropriate storage conditions (8). Most cases of corrosive substance ingestion involve boys under 5 years of age (2,7,8). Similarly, in our study, the mean age of the patients was 41 months (i.e. 3.4 years) and the male/female ratio was 1.34. Several Turkish studies have reported that corrosive substance ingestion constitutes 3.3%–28.1% of childhood intoxications (9). It has been reported that every year, approximately 5000 incidences of accidental corrosive substance ingestion occur in children under 5 years of age in the United States (3), while 1000–20,000

Table 1. The first endoscopic findings of the cases, listed according to the corrosive substances.

				Endoscopic findings of the cases at first inspection									
				Esophageal findings					Gastric findings				
Corrosive substance	pH	n	%	Normal	Grade I	Grade IIA	Grade IIB	Grade IIIA	Normal	Hyperemia	Superficial erosion	Ulcer and/or hemorrhage	Necrosis
Oil remover (sodium hydroxide)	Alkali	22	21.4	10	0	4	3	5	15	2	1	3	1
Drain cleaner (hydrochloric acid)	Acid	19	18.4	6	1	7	4	1	8	1	2	8	0
Bleach (sodium hypochlorite)	Alkali	17	16.5	8	8	1	0	0	16	0	1	0	0
Toilet drain cleaner (sulfuric + hydrochloric acid)	Acid	14	13.6	5	3	2	3	1	7	2	3	2	0
Stain remover (sodium hydroxide)	Alkali	4	3.9	3	0	1	0	0	3	0	1	0	0
Hair bleach (hydrogen peroxide)	Alkali	4	3.9	4	0	0	0	0	2	0	0	2	0
Drain cleaner (sodium hydroxide)	Alkali	4	3.9	1	1	0	1	1	1	1	0	2	0
Oven cleaner (sodium hydroxide)	Alkali	4	3.9	4	0	0	0	0	4	0	0	0	0
Polisher for dishwasher (citric acid)	Acid	3	2.9	1	2	0	0	0	3	0	0	0	0
Disinfectant (potassium permanganate)	Alkali	3	2.9	1	2	0	0	0	3	0	0	0	0
Battery juice (sulfuric acid)	Acid	2	1.9	0	0	0	2	0	1	0	0	1	0
Formaldehyde	Neutral	1	1	0	0	1	0	0	0	0	1	0	0
Pure vinegar (acetic acid)	Acid	1	1	0	0	0	1	0	0	0	0	1	0
Analgesic for toothache (phenol + chlorobutanol)	Acid	1	1	0	0	1	0	0	1	0	0	0	0
Household cleaner (ammonia)	Alkali	1	1	1	0	0	0	0	1	0	0	0	0
Wood polisher (ammonia + sodium hydroxide)	Alkali	1	1	0	1	0	0	0	1	0	0	0	0
Disinfectant (benzalkonium chloride)	Alkali	1	1	0	1	0	0	0	1	0	0	0	0
Paint thinner	Acid	1	1	1	0	0	0	0	0	0	0	1	0
Total, n (%)		103	100	45 (43.7)	19 (18.4)	17 (16.5)	14 (13.6)	8 (7.8)	67 (65)	6 (5.8)	9 (8.7)	20 (19.4)	1 (1)

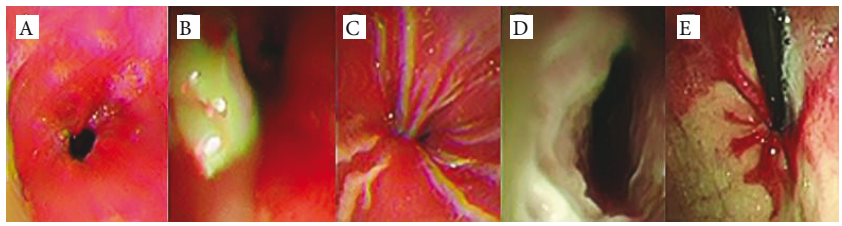

Figure 1. Endoscopic views of the patients: A) Grade I esophagitis, B) Grade IIA esophagitis, C) Grade IIB esophagitis, D) Grade IIIA esophagitis, E) gastric hemorrhagic ulcer.

Table 2. The locations of the esophageal and gastric lesions, according to the ingestion of alkaline or acidic corrosive substances.

The locations of the macroscopic lesions	Acidic substance ingestion, n	Alkaline substance ingestion, n	P
Only esophagus	14	20	>0.05
Only stomach	7	5	>0.05
Both esophagus and stomach	14	9	>0.05

cases of corrosive substance ingestion have been suggested to occur in industrialized countries each year (8). We did not find any data on the frequency of corrosive substance ingestion in Turkey. Although most cases of corrosive substance ingestion are accidental, they can also occur for the purpose of suicide, as shown in our study sample at a rate of 1.9%.

Substances that cause corrosive damage in children are mostly strong alkaline or acidic agents. Their liquid forms lead to more severe damage than their solid forms (8). In alkaline conditions, the pH of these substances can increase to up to 12, while acidic conditions can lead to pH decreases down to 2. Alternatively, the pH may remain neutral, around 7 (3). Recently, most cases of corrosive substance ingestion have occurred due to alkaline agents, especially in Western countries (2,10). In our study, too, most of the corrosive substances were alkaline (59.2%), and the rest had acidic or neutral characters (39.8% and 1%, respectively), similar to the values in the literature.

Detergents, soaps, shampoos, and destructive cleaners used for house cleaning, which do not contain bleach, are nontoxic and do not cause esophageal injuries (7). Dishwasher detergents, however, can cause esophageal injuries due to their additional alkaline character from the addition of sodium carbonate, sodium silicate, and sodium tripolyphosphate. Softeners contain cationic substances, which are more toxic than anionics and nonionics. If the cationic active substance rate is above 7.5%, the softener is capable of causing injury. Polishers used in dishwashers contain citric acid and do not cause very severe esophageal injuries (7). Other caustic cleaning substances include bleaches (sodium hypochlorite), drain cleaners (sodium hydroxide), and toilet/bath cleaners (sulfuric acid, hydrochloric acid, potassium hydroxide) (7,10). These are strong alkaline and acidic substances that can lead to severe tissue damage upon contact. Ammonia, which is frequently used for house cleaning, can not only cause caustic esophageal damage but can also lead to chemical pneumonia and pulmonary edema (8). Liquid bleaches (sodium hypochlorite) can demonstrate mild corrosive effects depending on their concentrations, amounts, and exposure times, but they do not cause tissue necrosis (7,8). Still, contraband bleach products having greater alkaline features and more additives could aggravate the severity of the corrosive damage. Most of the corrosive substances ingested by the patients evaluated in our study contained sodium hydroxide, hydrochloric acid, sodium hypochlorite, or sulfuric acid. Patients who ingest potassium permanganate are rarely reported (11). In our series, 3 patients had ingested potassium permanganate. Two of them suffered Grade I esophagitis. Other rare substances used as analgesics for toothaches, including phenol and chlorobutanol, caused Grade IIA esophagitis. In the literature, we did not encounter a patient with gastric outlet obstruction due to paint thinner ingestion. An interesting case was observed when a patient was admitted to our clinic with complaints of vomiting. He had a history of paint thinner ingestion 30 days previously.

Factors other than the pH of the agent responsible for the severity of the damage to the pharyngoesophageal passage include the amount, exposure time, and physical condition of the corrosive substance (liquid or solid form). In the esophagus, the cricopharyngeal junction, which is the region compressed by the aorta and left main bronchus,

and the lower esophageal sphincter are the regions at highest risk for caustic injuries and their complications (3).

The pathophysiological processes due to the ingestion of alkaline and acidic substances vary. Alkaline substances penetrate deep (to the layer of the muscularis propria), leading to mucosal lichenification followed by submucosal damage (3,5,12). In studies carried out on experimental animals, hemorrhage, thrombosis, evident inflammatory response, and edema were observed during the first 24 h following the ingestion of sodium hydroxide after lichenification in the epithelium or submucosal layer (8). As soon as alkaline corrosives are exposed to lipids through saponification, they are diffused deeply throughout the tissues, leading to edema and tissue loss. The only way to abort this reaction is by the neutralization of the material by the tissue itself. Because the alkaline substances taste relatively normal, they tend to be ingested in higher volumes, leading to even more severe potential damage. Acidic substances having worse and more bitter tastes generate coagulative necrosis on the mucosa; the formation of this scar tissue prevents further penetration by the agent and spreading of the damage. Damaged tissues facilitate bacterial infections. Approximately 48 h later, the esophageal wall begins to weaken due to fibroblast proliferation and collagen synthesis, which peak around the first to third weeks. The healing phase is usually completed during the fourth to sixth weeks with the formation of fibrosis and strictures (3,5). Respiratory distress, esophageal perforation, mediastinitis, and peritonitis are among the severe complications that accompany the periesophageal inflammation in the early phase (12).

While corrosive alkaline substances primarily lead to severe esophageal injuries, strong acidic substances can cause injuries to the stomach and even the duodenum (12). In different studies, the incidence of esophageal lesions has been reported as 34%–80% and the incidence of gastric lesions as 17%–62.5% after corrosive substance ingestion (1,2,11,13). In our study, the esophageal lesion rate was 56.3%, while the gastric lesion rate was 35%. Lesions that can be observed during endoscopic inspections, such as hyperemia, erosion, or ulcers in stomach, may be older lesions. This fact may have contributed to the percentage of lesions in our study.

The most common symptoms after the ingestion of corrosive substances are vomiting, dysphagia, hypersalivation, epigastric pain, and refusal to eat. Hyperemia, ulceration, and fragile white-colored membranes can be observed on the mouth, buccal mucosa, pharynx, tongue, uvula, and tonsils (8). Although a relationship has been reported among certain findings (14), such as hypersalivation, wounds on the mouth, leukocytosis, and the severity of the esophageal damage, these findings do not indicate that an endoscopic procedure is not necessary if those findings and symptoms are not present (8). Gupta et al. (15) suggested that endoscopic procedures were unnecessary in asymptomatic patients because their endoscopic examinations were normal. However, in a larger study, Gaudreault et al. (16) stated that signs and/or symptoms do not adequately predict the presence or severity of an esophageal lesion. Betalli et al. (17) proposed that the incidence of patients with third-degree lesions without any early symptoms and/or signs is very low, and endoscopy could be avoided. However, in our study, a patient who developed esophageal strictures only had Grade IIB corrosive esophagitis. Therefore, in our opinion, endoscopic evaluation is useful and required in corrosive substance ingestions to detect the degree of caustic damage to better plan the treatment, instead of waiting for the appearance of symptoms or complications.

Flexible fiber optic pediatric endoscopy is currently the most accurate and reliable device available to define esophageal and gastric lesions. Additionally, it is helpful to determine optimal therapy for the patient and to refer the patient to surgery in the early period if necessary (18,19). Endoscopic examination must be performed between 12 and 36 h after the ingestion, or suspected ingestion, of the corrosive substance (8).

Patients who ingested corrosive substances must not be forced to vomit and must not drink fluids orally, not even to neutralize the substance. The face, mouth, and buccal mucosa can be washed to extract the mucosal residues of the substance (8). Nasogastric tube application and gastric lavage are contraindicated (1,8). While patients who did not demonstrate mucosal injuries upon endoscopic examination can be discharged immediately, patients who had mild mucosal damage must be followed for 24–48 h. Patients with more severe damage must be followed and treated in the hospital for a longer time. Treatments include cessation of all oral intake, parenteral nutrition support, the use of wide-spectrum antibiotics, and intravenous antacid therapy (12). If oral nutrition is started, a liquid diet must be given for the first several days (20). H_2 receptor blockers and proton pump inhibitors have been used to either prevent or treat gastrointestinal ulcers for a long time, even though no studies have been performed on their use for the treatment of caustic damage. Only one study (21) proposed that omeprazole may be efficient for treating corrosive burn injuries to the esophagus.

Although corticosteroids have commonly been used for treatment previously, their use remains controversial due to conflicting reported results in their prevention of strictures (3,8,18,20). In a systematic analysis of 328 patients with Grade II esophagitis, 30/244 patients who received steroids and 16/84 patients who did not receive steroids developed strictures (22). This difference was not

statistically significant. In another analysis (23), 35.1% of 305 patients treated with corticosteroids developed strictures, whereas 33.3% of the 267 patients without corticosteroid treatment developed strictures. These data suggest that systemic corticosteroids are not beneficial for Grade II or more severe corrosive esophageal burns. Furthermore, gastric ulcers, esophageal mycotic infections, osteoporosis, and secondary bacterial infections are complications related to corticosteroid use (3,8). We preferred not to use corticosteroids to treat our patients. Corticosteroids are mainly useful in cases of an affected larynx or a developed edema on upper respiratory tracts (3).

Sequelae due to caustic injuries include strictures, achalasia, brachioesophagus, gastroesophageal reflux, and the development of a malignancy in the later period (8). Pyloric obstruction can develop after gastric lesions, especially due to the ingestion of acidic agents after 3–7 weeks (8,10,24,25). In our series, one of the patients who had a history of acidic agent (thinner) ingestion developed a gastric outlet stricture. Isolated gastric strictures are relatively rare and arise essentially due to acid ingestion (24,26). The reported rate of esophageal stricture development is 9%–18% (8). Most patients who developed strictures were those with Grade III esophagitis at their first endoscopic examinations. Strictures due to acute inflammation occurred within the first 21 days. However, complete stricture development takes 30–45 days (8). In our series, esophageal strictures had developed in 4.9% of the cases. Grade IIIA esophagitis was present in 4 cases and Grade IIB esophagitis in 1 upon the first endoscopic examinations.

In conclusion, in Turkey, corrosive substances that are used as household cleaning supplies are freely available either in marked commercial or unmarked contraband forms; most of these products are sold in liquid or crystalloid forms. In most of these cases, corrosive agents were present in the home in locations that the children were able to reach. Many of these agents are distributed in pots that look like water or beverage bottles. Therefore, efficient measures must be taken. When corrosive substances are ingested, an endoscopic examination within 12–24 h is useful for grading the injuries and providing important information when planning treatment and estimating the prognosis for both short- and long-term complications.

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