

Evaluation of 968 children with corrosive substance ingestion

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Abstract

Background and Aims: The aim of the study was to evaluate the etiology, treatment, and prognosis in children who had presented at our clinic with corrosive substance ingestion and comparison of our results with the literature. **Materials and Methods:** The patients were put on nil by mouth and broad-spectrum antibiotics were administered. Oral fluids were started for patients whose intraoral lesions resolved and who could swallow their saliva. Steroids were not given, a nasogastric catheter was not placed, and early endoscopy was not used. **Results:** A total of 968 children presented at our clinic for corrosive substance ingestion during the 22-year period. The stricture development rate was 13.5%. Alkali substance ingestion caused a stricture development rate of 23%. A total of 54 patients required 1–52 sessions (mean 15 ± 12) of dilatation. **Conclusion:** We do not perform early endoscopy, administer steroids, or place a nasogastric catheter at our clinic for patients who had ingested a corrosive substance. This approach has provided results similar to other series. We feel that determining the burn with early esophagoscopy when factors that prevent or decrease the development of corrosive strictures will be very important.

Keywords: Caustic ingestion, corrosive ingestion, esophageal stricture, esophagoscopy

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Introduction

Childhood corrosive substance ingestion is frequently encountered. It can cause esophagus perforation and mediastinitis-related death in the acute stage and esophagus strictures that require a difficult and long-term treatment in the late stage. A standard treatment has not been determined despite many studies on the treatment of corrosive esophagus strictures, and many clinics use their own treatment approach. Although reasonable success has been achieved with corrosive stricture treatment, the best method is obviously preventing corrosive substance ingestion with preventive healthcare service. We evaluated the etiology, treatment, and prognosis of patients who had presented at our clinic with corrosive substance ingestion in this study.

Materials and Methods

A total of 968 patients who had presented at our clinic for corrosive substance ingestion between 1990 and 2012 were retrospectively evaluated. The patients used to undergo routine esophagoscopy within the first 48 h at the early years of this period, but this was later discontinued.

Patients presenting for corrosive substance ingestion were hospitalized and put on nil by mouth. Broad-spectrum antibiotics were administered. Steroids

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were not used in any patient and nasogastric catheters were not placed. Patients whose oral lesions and general condition recovered and who could swallow their saliva were started oral fluids, usually within 1–12 days. Patients with no complications were discharged on a soft diet and told to come back on the 3rd week for an esophagogram.

Patients who had a stricture on the esophagogastroduodenal X-ray obtained on the 3rd week after corrosive substance ingestion were put in the dilatation program. The dilatation procedure was performed antegradely with a guidewire under general anesthesia and patients with no problems discharged the same day. A gastrostomy was opened if antegrade dilatation was unsuccessful. The gastrostomy was used both for feeding and retrograde dilatation.

Dilatations were once every week or 2 weeks depending on the severity of the stricture at the beginning and the intervals were gradually increased to 3–4 weeks depending on the clinical response. The procedure was stopped if minimal bleeding was encountered during the dilatation. A larger dilator was used once two dilatation sessions with a particular dilator size were uneventful.

Once the dilator size suitable for the patient was reached, the dilatation intervals were gradually increased to 6–8 weeks and the number of sessions was arranged according to the clinical course. Patients who completed their dilatation program were continued to be monitored at suitable intervals.

The “independent samples *t*-test” was used as the statistical method. $P < 0.05$ was considered significant.

Results

A total of 968 children aged 10 days to 17 years (mean 3.9 ± 3.1 years) who had ingested a corrosive substance presented at our clinic during the 20-year period. All except two of the corrosive substance ingestion episodes were accidental. The gender distribution was 58% male and 42% female. The corrosive substance was alkali in 75%, acid in 18%, other 5%, whereas 2% did not know the characteristics of the substance. The most commonly ingested corrosive substance was bleach, followed by lipid dissolver, limescale dissolver, and hydrochloric acid. Strictures did not develop in any patient who had ingested bleach. Stricture development due to corrosive substance ingestion was seen most commonly with caustic soda, ammonium chloride, and lipid dissolver ingestion. The most common cause of stricture development was lipid dissolvers. Caustic soda contains a high concentration (46%) of NaOH and has

marked corrosive effects. The chemicals sold as lipid dissolver in markets are generally products that contain NaOH at 5–15% concentration and are used for oven cleaning. These were found to be the most important cause of strictures because they are both used often and are highly corrosive. Chemicals containing NaOH and KOH, used to open blocked pipes, were also found to be the common causes of corrosive strictures [Table 1].

Corrosive strictures did not develop in any patient due to bleach ingestion. We therefore excluded this group of patients and found that esophagus strictures developed in 69 (13.6%) of the 508 children who had ingested corrosive substances.

The rate of corrosive stricture development was 8.2% and 2.9%, respectively, in children who had ingested alkali or acid substances ($P < 0.05$). The stricture development rate due to alkali substance ingestion was 23% when bleach was excluded ($P < 0.05$).

Vomiting was reported by 52% of the patients following corrosive substance ingestion. The stricture development

Table 1: Corrosive substances ingested and stricture development rates

| Substance | n | Percentage | Stricture (n) | Stricture % |
|---|-----|------------|---------------|-------------|
| Bleach* (NaOCl) | 460 | 47.5 | 0 | 0.0 |
| Lipid dissolver* (NaOH) | 155 | 16.0 | 48 | 31.0 |
| Limescale dissolver** (HCL, HNO ₃ , H ₃ PO ₄) | 80 | 8.3 | 2 | 2.5 |
| Hydrochloric acid** (HCl) | 51 | 5.3 | 1 | 2.0 |
| Pipe opener* (NaOH, KOH) | 45 | 4.6 | 5 | 11.1 |
| Detergent* | 30 | 3.1 | 1 | 3.3 |
| Thinner*** | 21 | 2.2 | 0 | 0.0 |
| Unknown | 16 | 1.7 | 5 | 31.3 |
| Dishwasher polisher** (citric acid H ₃ PO ₄) | 16 | 1.7 | 0 | 0.0 |
| Surface cleaner* | 14 | 1.4 | 1 | 7.1 |
| Potassium permanganate (oxidant, disinfectant) | 10 | 1.0 | 0 | 0.0 |
| Spot remover* | 9 | 0.9 | 1 | 11.1 |
| Benzalkonium chloride (disinfectant) | 11 | 1.1 | 0 | 0.0 |
| Adhesive | 8 | 0.8 | 0 | 0.0 |
| Carpet cleaner** | 7 | 0.7 | 0 | 0.0 |
| Wart solution** (salicylic acid) | 8 | 0.8 | 1 | 12.5 |
| Hydrogen peroxide (disinfectant, whitener) | 11 | 1.1 | 0 | 0.0 |
| Caustic* (NaOH) | 4 | 0.4 | 3 | 75.0 |
| Laundry softener** | 3 | 0.3 | 0 | 0.0 |
| Sulfuric acid** | 2 | 0.2 | 0 | 0.0 |
| Kerosene*** | 2 | 0.2 | 0 | 0.0 |
| Phenol** | 1 | 0.1 | 0 | 0.0 |
| Marble polisher** | 1 | 0.1 | 0 | 0.0 |
| Ammonium chloride** | 2 | 0.2 | 1 | 50.0 |
| Rust dissolver** | 1 | 0.1 | 0 | 0.0 |
| Total | 968 | | 69 | |

*Alkali, **Acid, ***Solvent

rate was 18.3% and 2.5%, respectively, in patients who had vomited and those who had not. The difference was statistically significant ($P < 0.05$).

Dilution had been attempted by the family with liquids such as water, milk, and yoghurt before bringing the child to the hospital in 47% of the cases. The stricture development rate was 11.7% and 8%, respectively, in children where an attempt had been made to dilute the substance or not ($P > 0.05$).

The lips, oral mucosa, tongue, and oropharynx were evaluated for burns in the children presenting at our clinic for corrosive substance ingestion. Burns were found in 25% whereas no pathology was detected on physical examination in 75%. The stricture development rate in these groups was, respectively, 18.5% and 0.9% ($P < 0.05$). Stricture development ratios according to age groups are demonstrated in Figure 1. Although stricture development ratios regarding age groups look distinct, the difference is not statistically significant ($P = 0.227$).

A total of 69 patients were placed in the dilatation program and 54 completed this treatment (78.2%). The dilatation period was 3 weeks to 39 months (mean 44 ± 37 weeks). The patients underwent 15 ± 12 dilatations at 1–52 sessions [Table 2]. Patients who were removed from the dilatation program were followed up for 1 month to 6 years (mean 23 ± 18 months). Five of these patients presented 12–72 months after the last dilatation with mild sticking during swallowing. Minimal

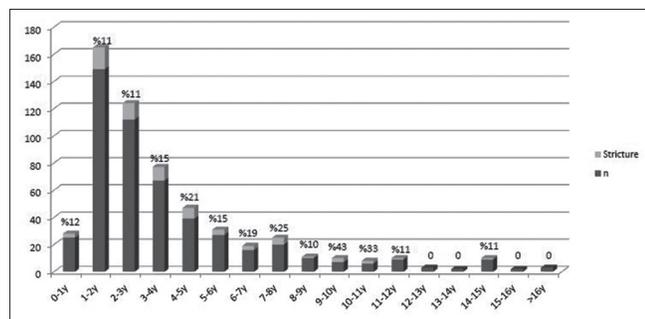


Figure 1: Stricture development ratios according to age groups

Table 2: Number of dilatations for the patients

| Number of dilatations | Number of patients | Percentage |
|-----------------------|--------------------|------------|
| 1-5 | 14 | 26 |
| 6-10 | 11 | 20 |
| 11-15 | 12 | 22 |
| 16-20 | 5 | 9 |
| 21-30 | 5 | 9 |
| >30 | 7 | 13 |
| Total | 54 | 100 |

narrowing was seen on the barium esophagogram and foreign bodies were sticking at the narrowed area in 3 cases. These patients underwent foreign body extraction and dilatation under general anesthesia and had no further problems.

Four patients (5.7%) underwent colonic transposition at another center due to strictures not responding to dilatation. The dilatation treatment of 2 patients is also continuing at another center. Four patients did not come for follow-up while their dilatations were continuing without complication. The dilatation treatment of 5 patients is continuing.

Only one patient developed esophagus perforation during the dilatation sessions (1.4%). A gastrostomy was opened and tube thoracostomy performed, and the perforation area closed spontaneously within 3 weeks.

Gastric outlet obstruction developed in four patients (0.8%) during the study period. Two patients underwent gastroduodenostomy, one gastrojejunostomy + truncal vagotomy, and one antrectomy + gastrojejunostomy. The causative substance was acid in three patients and alkali in one. There were no complications during follow-up.

No mortality was observed in our series.

Discussion

Esophageal strictures due to accidental ingestion of corrosive substances are a serious public health problem. This can largely be avoided by inspecting the production of substances containing corrosive substances, selling them in childproof packages, placing visible labels that can easily be read, and warning the public.

Corrosive substance ingestion can be life-threatening with esophagus-stomach perforation and large vessel penetration in the early stage while it may end in strictures requiring long and complicated treatment in the long-term.^[1] Acid and alkali substances can both cause corrosive esophagus strictures, but most are due to alkali substance ingestion, as in our series.^[2-4] Alkali substances cause liquefaction necrosis that can penetrate deeply whereas acid substances cause self-limiting coagulation necrosis. Alkali substances, therefore, cause strictures more often.^[5]

Treatment attempts in the acute stage for children who have ingested corrosive substances are aimed at

preventing the development of strictures. Steroid usage is controversial. Steroids are usually found not to be of value for first and third degree esophageal burns whereas some benefit is expected in second degree burns.^[4,6] Abandoning steroid usage has been suggested as it may be harmful while possibly lacking any benefit.^[7] Steroids were not used for any of the patients in our series. The rate of stricture development in our series was 13.6% when those ingesting bleach were excluded and this rate is comparable to those reported in patients using steroid. Our rate of stricture development was only 7.1% when we include those who had ingested bleach as well, as in many series.

Some series have followed up the patients by placing a nasogastric catheter for 1–6 weeks in the acute stage. The aim of this procedure is to prevent the esophagus mucosa from contacting food. Preventing this contact is thought to decrease bacterial colonization and inflammatory reaction and therefore stricture development. It has also been reported to prevent complete obliteration of the esophageal lumen.^[1,3,8,9] We did not place a nasogastric catheter to the patients in our series.

The stricture development rate for our patients was markedly higher in those who had vomited after corrosive substance abuse compared to those who had not (18.3% vs. 2.5%). Contact of the damaged esophageal mucosa with gastric acid may be increasing the damage. We did not find a marked difference in stricture development rate between patients who had undergone dilution with liquids such as water, milk, and yoghurt and those who had not, but one must keep in mind that the dilution procedure may also provoke vomiting.

Esophagoscopy within the first 48 h is a common procedure in patients who have ingested a corrosive substance. Some centers perform esophagoscopy in all patients who have ingested a corrosive substance,^[1,2,4,9] whereas others may only use it for patients who have ingested strong corrosive substances or have marked symptoms.^[10] Gupta *et al.* have reported that they did not find burns in the esophagus in any asymptomatic patient whereas all patients with severe esophagus burn suffered from symptoms (salivation, being unable to swallow, vomiting, stridor, etc.). The authors have suggested not performing esophagoscopy in asymptomatic patients.^[11] Betalli *et al.* have reported from their multicenter study that endoscopy is not mandatory in asymptomatic patients but should be performed in all symptomatic patients.^[9] We used

to perform esophagoscopy routinely for all patients presenting with corrosive substance ingestion at our clinic. However, we found that the esophagoscopy findings did not change our clinical approach to the patients and stopped performing the procedure 7 years ago. This approach did not have a negative effect on the prognosis of the patients. We also followed up patients who had ingested bleach, who made up 47% of our series for more than 10 years without hospitalization and did not observe any complications in any of them.

Bicakci *et al.* have treated their patients with a so-called “minimal invasive approach” where no early endoscopy, systemic steroids, or nasogastric catheter was used. They report good results with this approach that is very similar to ours.^[12]

Conclusion

We were unable to find any conclusively beneficial reported method to prevent or decrease stricture formation in children who had ingested corrosive substances. On the other hand, since results of minimal invasive approach regarding outcomes of our current study are in concurrence with the literature, we believe that this approach is reasonable.

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Conflicts of interest

There are no conflicts of interest.

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