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Main topic

Caustic ingestion in childhood: current treatment possibilities and their complications

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Abstract. In a review of 15 pediatric patients who had ingested caustic substances, the authors describe the diagnostic and therapeutic procedures to be followed as well as the complications that may occur with their use. The cases reported include 1 esophageal rupture caused by balloon dilatation and 1 recurrent stenosis treated with a silastic tutor.

Key words: Caustic ingestion – Esophageal stenosis – Esophageal dilatation

Introduction

Accidental ingestion of caustic substances is a common event in childhood: in the United States alone, more than 5,000 cases are reported annually in children under 5 years [10]. During adolescence, ingestion of these substances in some cases represents an attempt at suicide.

The use of endoscopy in pediatric patients has considerably improved the prognosis for pathology caused by caustic ingestion [2, 14, 18]. With esophagoscopy, it is possible to make a prompt and precise evaluation of the degree of damage, initiate appropriate therapy, and monitor the sequelae of the lesions. The endoscopic technique of balloon dilatation [11, 16, 18] has almost eliminated the need for esophagoplasty, which, in addition to being a rather complicated and traumatic surgical procedure, is always associated with some degree of dysfunctional sequelae.

This report describes the diagnostic work-up and therapeutic management of 15 children seen in our medical center for caustic ingestion. Some of the complications that can arise during treatment are discussed and possible approaches for their resolution are presented.

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Patients and methods

Between April 1986 and August 1991, 15 children were treated in the Pediatric Surgery Department of the Policlinico "A. Gemelli" in Rome for ingestion of caustic substances. Their ages ranged from 2 to 9 years with a mean of 3.3 years. The male/female ratio was 3:1. Twelve (80%) of the children had ingested alkalies (sodium hydroxide, detergents, ammonia) and the remaining 3 had consumed acids (sodium hypochlorite).

All of the patients were subjected to emergency esophagogastroduodenoscopy (EGDS), in most cases within the first 12 to 24 h following ingestion. This allowed us to document any esophageal and/or gastric lesions and to plan our treatment on the basis of their severity. Thirteen (87%) of the children presented with lesions of the oral mucosa, but esophagitis was present in only 10. The esophageal lesions varied in severity: 3 children had grade I burns, 2 were classified as grade II, and 5 had grade III lesions (Fig. 1 a). In one patient there was involvement of the gastric antrum. None of the children suffered duodenal damage.

Only those patients with documented evidence of grade II or III esophagitis were treated. Those with grade I lesions wer discharged from the hospital after 24–48 h of clinical observation.

Results

Our treatment protocol, which is advocated by most authors [2, 7, 8, 10, 18, 20], provides for the use of broad-spectrum antibiotics, high-potency corticosteroid anti-inflammatory agents, and H_2 antagonists. Suspension of feedings was not necessary in any of the children. Those with milder grades of esophagitis were given cold liquids by mouth. In the more severe cases a nasogastric tube was placed to avoid trauma to the esophageal mucosa during swallowing and to maintain patency of the lumen for later dilatation. None of the children required total parenteral nutrition.

Five of the children with grade III esophageal lesions (2 of whom had been referred from other hospitals) developed stenoses. These lesions were treated with endoscopically guided balloon dilatations beginning on the 20th day post-ingestion and continuing at 10-day intervals (Fig. 1 b and 1 c). In 3 of these 5 children, complete resolution of the stenosis was achieved with a mean of nine dilatations.

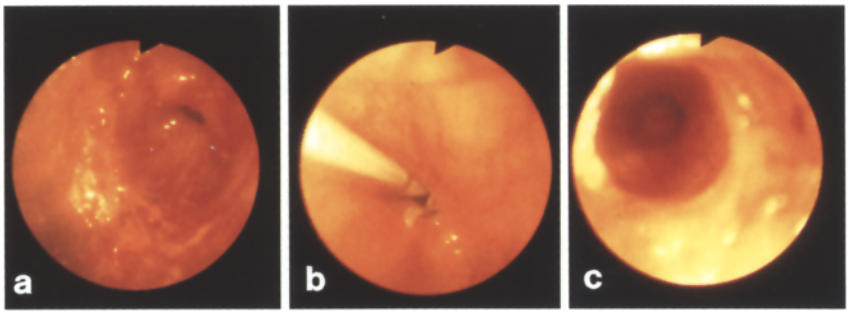

Fig. 1. a Endoscopic view of serrate stenosis in proximal third of esophagus following grade III burns. **b** Deflated balloon catheter positioned at level of stricture. **c** Endoscopic view of stenotic area after dilatation

The 4th patient, a 6-year-old boy, sustained a perforation of the esophagus by an oversized catheter during the 6th dilatation session. The perforation occurred in the distal thoracic portion of the esophagus, although the stenotic lesions themselves were high, and was accompanied by violent epigastric pain, dyspnea, subcutaneous emphysema, and fever; chest radiographs showed signs of a pneumomediastinum. An esophagogram with gastrografin was performed to confirm the diagnosis, and we then performed an emergency thoracotomy through the right 7th intercostal space. The small lesion in the anterior wall of the esophagus, approximately 3 cm above the diaphragm, was sutured, and paralesional and pleural drainage tubes were left in place. One month later, when the perforation had healed completely, dilatation was resumed using smaller-caliber catheters and lower pressures. After 6 more sessions, the stenosis was completely resolved.

The 5th patient, also 6 years old, had undergone retrograde esophageal dilatation with catheters of increasing diameters in another hospital. The catheters were being passed through a gastrostomy that had been created for feeding. In spite of several months of treatment, the strictures had always recurred. After performing 10 more balloon dilatations without achieving any lasting degree of patency, we resorted to the use of a silastic transesophageal tutor. The tutor was left in place for 21 days, and the results achieved were excellent and lasting.

There was no long-term follow-up of the children who had presented with grade I esophageal lesions. All of the other children were submitted to a repeat esophagogram 2 years after the caustic damage had occurred. Stabilization of the scarring process does not, in fact, occur prior to this time. All of the lesions were found to have healed completely and have remained asymptomatic.

Discussion

The caustic substances ingested by children are in most cases alkaline; ingestion of acids is rarer and causes esophageal lesions in only 6%–20% of all cases [14]. Acids cause superficial coagulation of the epithelial proteins that does not often extend into the deeper layers of the esophagus. Their effects are also partially neutralized by the mildly alkaline esophageal pH. The damage caused by ingestion of acid substances is more often seen in the gastric antrum. This is partially due to the fact that the contact between the acid and the gastric mucosa is longer than that in the esophagus, but the absence of a buffer system at this level also contributes to the damage.

Esophageal damage is more common and more severe with the ingestion of alkaline substances, especially sodium and potassium hydroxide, which are present in many detergent products. Alkaline substances induce erythema and edema of the esophageal mucosa (grade I burns) that can rapidly extend to the submucosal (grade II) or muscular layers (grade III). In some cases the burns are so severe that they perforate the esophagus and the alkalies leak out to cause damage to the periesophageal structures with mediastinitis and occasional formation of tracheoesophageal fistulas. The lesions are usually located in areas of physiological narrowing of the esophagus. If large quantities of alkalies are swallowed there may be duodenal damage, but the stomach is usually spared [17].

Most children who have ingested caustic substances present with visible burns in the oral cavity, but only 50% have esophageal lesions as well [10, 20]. In general, esophageal involvement is more likely when the oral lesions are more severe, but esophageal burns can also be found when there are no oral lesions whatsoever [20]. Symptoms (nausea, anorexia, vomiting, dysphagia, abdominal pain, fever) and hematologic alterations (leukocytosis) have proved to be poor indices of esophageal or duodenal damage [6]. Esophagograms are also of little use in the acute phase: at this time they provide poor definition of the mucosal lesions, and the spastic reaction of the esophagus makes interpretation difficult.

For these reasons, endoscopy is essential in the initial evaluation of a child suspected of having ingested caustics. It allows the physician to confirm the diagnosis, evaluate the degree of damage, and initiate appropriate therapy promptly. Most authors [2, 7, 8, 10, 18, 20] agree that esophagogastroscopy should be performed with the patient under general anesthesia within the first 12–24 h after ingestion. After 24 h the risk of perforating the esophagus during this examination increases.

If EGDS shows only grade I burns, the patient should be observed for 24–48 h; no treatment is usually required. In patients with grade II or III lesions, it is essential that prophylactic antibiotic and steroid treatment be provided. The former is aimed at reducing the risk of pulmonary infections and preventing the complications associated with perforation. Ampicillin is generally used; the dosage and length of treatment vary according to different authors [2, 7, 10]. Steroids are usually started during the first 48 h and continued for 3–4 weeks; prednisone is commonly used at a dose of 2 mg/kg per day [9].

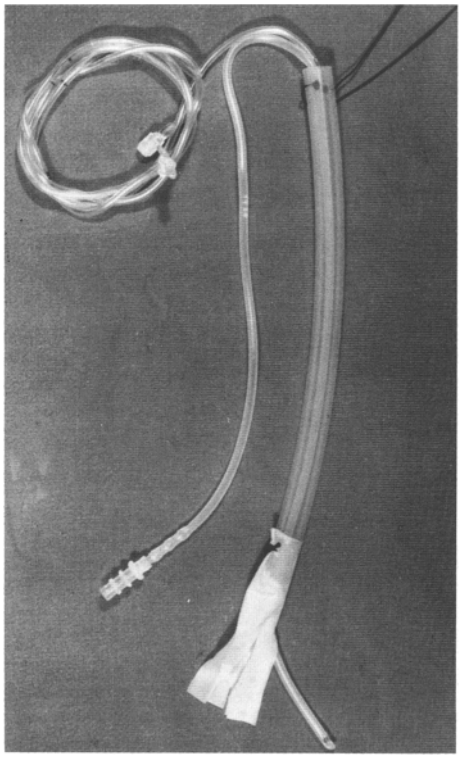

Fig. 2. Silastic transesophageal tutor

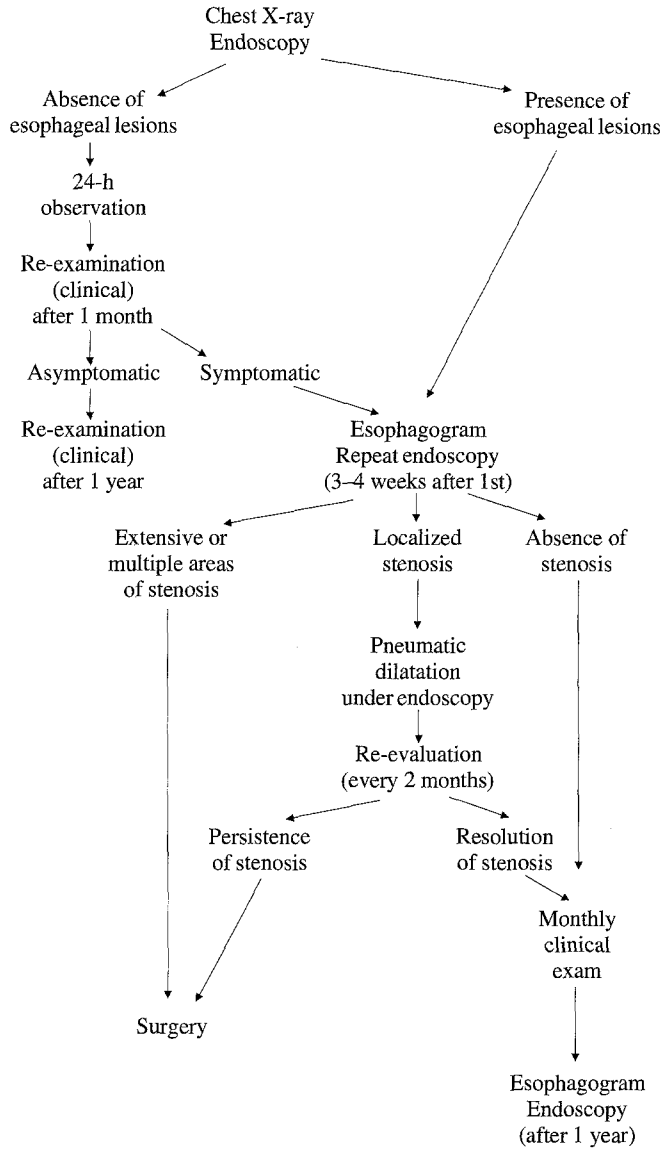

Fig. 3. Diagnostic work-up for caustic ingestion

A consensus has not yet been reached, but this approach seems to be effective in reducing the incidence of cicatricial stenosis in grade II burns; its effects in grade III lesions and burns involving more than two-thirds of the circumference of the esophagus are somewhat less satisfactory. The presence of ulcers, perforations, or major infections (pulmonary and/or systemic) represent absolute contraindications for the use of steroids.

Endoscopy has also provided new treatment possibilities for those cases in which stenosis develops. Balloon dilatation of these strictures is performed with a catheter equipped with an oval-shaped balloon along its terminal length. Under endoscopic visualization, the balloon portion of the catheter is positioned at the level of the stricture and normal saline is injected into its lumen to "inflate" the balloon to a pressure of 3–5 atm. Catheters are available with balloons of various diameters ranging from 8 to 30 mm. Diameters of 10–15 mm are generally used for pediatric patients.

Balloon dilatation is not indicated in cases of extensive stenosis and is not entirely free of complications. Esophageal perforation caused by balloon dilatation is a rare event occurring in from 1% to 5% of all cases [1, 4], most frequently in the anterolateral wall 3–4 cm above the diaphragm. In this area the muscle fibers of the esophageal wall intersect in such a way that a "locus minoris resistentiae" is created. The perforation is probably caused by a traction mechanism [13].

The occurrence of symptoms consistent with pneumomediastinum (retrosternal pain, fever, subcutaneous emphysema) requires an immediate chest radiograph, which will not only confirm the presence of a pneumomediastinum but may also reveal a pneumothorax. An esophagogram should then be performed with water-soluble contrast medium to locate the lesion itself.

The management of iatrogenic perforations of the esophagus is still an object of debate. Some authors [3, 19] advocate conservative treatment limited to antibiotic coverage and total parenteral nutrition; we agree with others [13] who insist that surgical repair of the perforation is preferable. In some cases the degree of patency that can be achieved with balloon dilatation is less than satisfactory. Before resorting to surgery in these cases, an attempt can be made to place a silastic tutor.

Figure 2 shows the tutor we created using a silastic tube. In the case described above, the length of the tutor was 16 cm; its external diameter was 1.1 cm and the internal diameter 0.9 cm. At its distal tip a fringed Penrose drain was attached to prevent gastroesophageal reflux. At the cranial end we attached a thin double-lumen tube that was used to clean the tutor and remove excess secretions. Two

silk sutures were also attached to the cranial extremity of the tutor and prevented the tutor from slipping down toward the stomach.

Following balloon dilatation during general anesthesia, the tutor was positioned with the aid of a pediatric bronchoscope inserted inside its lumen. The correctly positioned tube extended distally for 1–2 cm into the stomach; its cranial tip lay approximately 1 cm above the cricopharyngeal sphincter. The two silk sutures and the double-lumen tube mentioned above protruded from the child's nostrils, as did the proximal portion of a nasogastric tube that had been passed through the tutor lumen to administer feedings. The tutor was left in place for 20 days and was well tolerated by the patient. At the end of this period, it was removed by means of the silk sutures.

While the tutor was in place, antibiotic and steroid therapy were provided. The latter was initially administered at full dosage and then gradually decreased. Steroids were finally discontinued 1 week before removal of the tutor. This approach resulted in definitive resolution of the stenosis.

In conclusion, based on our experience and that described in the literature, we feel that the correct management of esophageal lesions provoked by ingestion of caustic substances should be based on the following points (Fig. 3):

1. Esophagogastroduodenoscopy under general anesthesia during the first 12–24 h after ingestion to confirm the existence of lesions and evaluate their severity [2, 7, 8, 18, 20].
2. Simple clinical observation of children with grade I lesions for 24–48 h without further treatment [2, 7, 8].
3. Immediate initiation of medical treatment (antibiotics, steroids, H_2 antagonists) and placement of a nasogastric tube for feeding in patients with grade II or III burns; repeat EGDS after 20 days to evaluate the lesions evolution and after 40 days to rule out the occurrence of stenosis [2, 7, 18].
4. Endoscopically guided balloon dilatation of any areas of stenosis beginning on the 20th post-ingestion day and repeated every 10–15 days until the stricture(s) have been eliminated [2, 5, 11, 12, 16, 18]; placement of a silastic esophageal tutor for 20–30 days in cases in whigh stenosis recurs after balloon dilatation [15, 21].
5. Esophagoplasty in the rare cases of extensive stenosis or those that do not respond to the dilatation approaches described above.

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