Caustic Ingestion and Foreign Bodies

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16.1 Introduction

Their homes and surroundings can be dangerous places for children, particularly with regard to the possibility of unintentional swallowing of foreign bodies or potentially dangerous liquids.

Children are naturally curious, exploring in and around their homes. They develop their senses by physically interacting with the things around them, touching and placing them in their mouth. As a result, each year millions of calls are made to poison control centers or pediatric emergency rooms after unintentional swallowing of foreign bodies or potentially dangerous liquids. Most of these accidents could have been prevented. This chapter focuses on caustic and various foreign body ingestions [1].

16.2 Caustic Injuries

16.2.1 Epidemiology of Caustic Injuries

Ingestions of corrosive substances, alkalis or acids, are common both in low- or high-income countries in spite of prevention measures to minimize the hazards of household products and laws for containers with child-resistant closures. The majority of ingestions occurs in children younger than 5 years and could be preventable [2–4]. Ingestion in children older than 5 years is suspect and in adolescents, mainly in girls, is usually intentional with larger volumes swallowed [2]. In addition, there might be an unknown number of cases of abuse.

The true prevalence of these injuries is unknown. According to the report on pediatric trauma done by the World Health Organization and the UNICEF, more than 120,000 children under 6 years old suffered caustic injuries in the United States in 2004 [1, 5]. While most exposures to household products result in mild poisoning, cleaning agents, strong alkalis, and acids can lead to severe tissue damages. In the pediatric group, 70–90 % of burns are related to alkali substances and 10–30 % to acids [2–4, 6, 7].

Chemicals around the house to which children may have access contribute significantly to unintentional poisonings in childhood, both in developed countries and in low-income countries. Only the substances differ. In developed countries exposures to cleaning agents, such as ammonia, bleach, dishwasher, and laundry detergents, are most common, especially the dishwasher tablets that are the most frequent household products involved in injuries [2–4]. Dishwasher detergents are highly corrosive substances causing potentially life-threatening injuries and severe morbidity [4, 7, 8]. In most countries dishwasher tablets are not included in the regulations

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for child-resistant closures. Another concern is that liquid household products such as soaps, liquid soaps, and dishwasher and laundry detergents are packaged to imitate food or have other attributes that appeal to children (smell, color). Due to new trends in food marketing, the frontier between food products and cosmetics has been blurred. In 2011, the Scientific Committee on Consumer Safety of the European Commission stated that "Products which, although not foodstuffs, possess a form, odor, color, appearance, packaging, volume or size, so that is likely that consumers, especially children, will confuse them with foodstuffs and in consequence place them in their mouths, or suck or ingest them, which might be dangerous and cause, for example, suffocation, poisoning, or the perforation or obstruction of the digestive tract" [9]. These recommendations are fairly followed, as prevention does not carry much weight compared with the expected benefits.

This last decade, new concentrated laundry "pods" or capsules appeared on markets and are associated with more severe accidents compared to classic laundry detergents. Detergent "pods" are small, single-use doses of concentrated detergent encased in a water-soluble membrane (polyvinyl alcohol) to dissolve in wash water and release the detergent. They are colorful designed like candies. Compared to classic laundry detergents, the chemical composition of laundry pods has a higher concentration of surfactants and ethoxylated alcohols and a higher viscosity and hydrotropic power. They are filled under pressure, so when the child places the capsule in his mouth and bites it or sucks on it, thus dissolving the water-soluble membrane, the content explodes before the eyes or in the mouth with subsequent ingestion and/or inhalation. A spectrum of clinical effects from minor to serious injuries, even deaths, was seen with ingestions, inhalations, ocular exposures, or combinations of them. In the USA, from 2012 to 2013, 17,230 children exposed to laundry detergent pods were reported to US poison control centers [8, 10]. In Italy, laundry detergent pods have become the most commonly ingested household product since becoming available in 2010 [11]. From 2012 to 2015, 34 reports from different countries (France, Canada, the USA, Italy, the UK) came to the same conclusions and warn clinicians, parents, and caregivers. They all conclude that measures should be taken to avoid ingestions of these products, but nothing has been done to date to regulate their use and composition. "These publications, although commendable for resulting in positive outcomes, also serve to highlight previously identified weaknesses in the NPDS surveillance system (USA, but other national agencies as well)" [12].

In developing or lower-income countries, sodium hypochlorite or sodium hydroxide (lye, caustic soda), used to make soap, as a bleaching agent, to manufacture textiles, for washing or chemical peeling of fruits and vegetables, for cocoa processing, for olive softening or blackening (also with potassium hydroxide), or to prepare "medicines," is left reachable for children often on the ground.

16.2.2 Prevention of Caustic Injuries

The most obvious risk factor for ingestion of a substance is its presence in the domestic environment, within the reach of children. Dispensing them in containers without child-resistant closures increases the risk of poisoning. The use of appropriate labeling ("skull and crossbones") serves as parents' warning but not to children who are unlikely to recognize the significance of these signs. Subsequently, the best prevention is to keep them out of their reach. Bathroom cabinets and kitchen cupboards or locked drawers appear to be the safest storage places. Unfortunately, in modern houses, the bad habit is to store harmful products in the locker under the sink instead of putting them in a high place. Safe packaging cannot compensate for unsafe storage.

In high-income countries, dangerous products are required by law to be distributed in child-resistant packaging, i.e., requiring several complex actions such as turning while pushing downwards or squeezing. The common standards for tests adopted in most countries require that at

least 85 % of children aged from 42 to 51 months must be unable to open the container within 5 min [13]. Child-resistant packaging is one of the best-documented successes in preventing the unintentional poisoning of children [1]. Unfortunately, rules for child-resistant packaging currently exist only in very few countries, such as Australia, Canada, New Zealand, the United States, and the European Union. Furthermore, no closure is perfect. Up to 20 % of children aged between 42 and 51 months may be able to overcome a child-resistant closure, and their parents are most often unaware [14].

An alternative approach is to lower the level of the toxicity or to make it repellent or uninteresting for children. Toxicity can be lowered by reformulation and many dangerous substances could be replaced. Color can play a role, as pink, purple, and yellow are attractive for children, while dark blue, violet, or brown-green are unappreciated colors. Children initially prefer sweet tastes and reject sour and bitter tastes. Therefore bittering agents have been used to prevent from ingestions and poisonings. The most commonly used aversive agent is the denatonium benzoate (Bitrex® or Aversion®), which has an unpleasant taste at very low concentrations unbearably bitter to most humans [15–17]. The problem with sodium hypochlorite or hydroxide is their lack of smell and their harmless appearance. The addition of <1 % ammoniac is enough to give them an unpleasant smell with a subsequent repellent effect on children, but unfortunately also on their mothers who will not buy such a product. Unfortunately there are no published data on the effectiveness of aversing agents in limiting the ingestion of household products.

In developing or lower-income countries, these dangerous products are freely bought on markets and diluted or transferred in beverage bottles at home where they are stored on the ground or in places reachable for children. As the manufacturing facilities are scarce, it could be cheap and easy to color sodium hypochlorite or hydroxide and to add ammoniac. During our numerous missions, we have frequently received interest from many governmental or nongovernmental authorities but without effects.

16.2.3 Physiopathology of Caustic Injuries

Both acids and bases can be defined as caustics, which cause significant tissue damages on contact with the esophagus. Most acids produce a coagulation necrosis by denaturing proteins, inducing a coating coagulum that protects the underlayers from deeper penetration. Bases induce more severe injuries known as liquefactive necrosis, i.e., the denaturation of proteins together with a saponification of fats, which penetrate deep through the esophageal wall and can go through it. The lesions are colonized by bacteria within 24–48 h worsening the tissue damages.

The severity of the damages is related to several factors, including the pH, the concentration, and the volume of the agent. The contact time is of little interest as a lesion occurs within a few seconds. The physical form of the agent plays a significant role: the ingestion of solid pellets results in prolonged local contact time with the esophagus, thus deeper localized burns, while liquids generate superficial but more extensive lesions. For this reason it is of major importance to refrain from drinking after pellet ingestion as it may induce both types of lesion. Vomitings – spontaneous or induced using emetic – worsen the lesions due to a repeated exposure.

Depending on the extent of burn, inflammation or necrosis may extend through the whole esophageal wall until perforation occurs either in the mediastinum with subsequent mediastinitis or in the trachea or bronchi. With the disappearance of the mucosa, the facing surfaces adhere to each other worsening the stenosis of the esophagus or occluding its lumen, moving toward a fistula. Like the skin, the long-term effect of caustic esophageal burns is a hypertrophic scarring process, which can result in stricture formation. Mucosal reepithelization is a slow process, usually not complete before 4–6 weeks. Not until a complete reepithelization, the inflammation continues, and granulation tissue comes to maturity when fibroblast proliferation replaces the submucosa and muscular layers, initiating strictures. Thus a stricture formation is detectable after 2

weeks and is definite by the fourth week. This is the best time to start dilatations. In a series of 80 pediatric patients, de Jong reports 29 % of early and late medical complications and 20 % that developed severe esophageal strictures requiring esophageal replacement in two-thirds of them [18]. Baskin reports 81 of which 16 % developed a stricture even in some cases of low grades [19].

If the muscular layers of the esophagus have been destroyed, they will not regenerate and be replaced by fibrous tissue. Even if the lumen has been kept open, the contraction waves will never overpass that point.

The caustic burn induces a shortening of the esophagus and a motility disorder resulting in reflux and poor esophageal clearance, which adds a peptic stenosis to a caustic one evidenced by histology (O. Reinberg, unpublished). For this reason all our patients under conservative treatment with dilatations receive proton pump inhibitors (PPI).

16.2.4 Diagnosis of Caustic Injuries

A suspicion of caustic ingestion requires a detailed questioning, asking for the nature of the product, its form, the amount, and the precise time of the injury. When receiving an emergency call, remind the parents or the caregivers to avoid drinking or eating (no emetic agent) and ask to bring the product and the packaging with them. This will help to identify the ingested substance and measure its pH.

There is a large variation of symptoms after caustic ingestion ranging from nothing to life-threatening conditions. Several studies have indicated that the clinical manifestations are poor predictors of the presence and the extent or depth of esophageal injury [3, 4, 18, 20]. Initial symptoms and clinical signs are mostly related to the edema. However, the presence of more than three symptoms or signs is associated with increased likelihood of esophageal injury [3]. Reversely, Gandreault wrote that 12 % of children with proven esophageal injuries had no significant esophageal or abdominal complaints [20]. Thus any suspected caustic ingestion should be referred to medical facilities to be investigated.

The most common symptoms are drooling, dysphagia, odynophagia, vomiting, and oral liquid refusal. Respiratory symptoms such as tachycardia, dyspnea, dysphonia, and stridor are evocating upper airway injury and may be seen immediately or delayed due to the progressive edema, but can be seen without airway involvement. Hematemesis is related to severe esophageal injuries, extensive or deep. Chest or abdominal pain and rigidity suggest profound injury and perforation of the esophagus or the stomach [3].

The oral cavity should be carefully inspected to look for lip swelling, tongue erythema, leukoplakia, or oral ulceration. Blind placement of a nasogastric tube should be avoided due to the increased risk of perforation.

Chest, lateral neck, and abdominal X-rays are systematically done to look for the presence of free air in the retropharynx, mediastinum, or peritoneum.

Contrast studies (UGI) are not helpful at early stages. They do not reveal mucosal injuries or overestimate the lesions showing mainly the edema. They represent a waste of time postponing the endoscopy. If done, the use of barium should be avoided in case of perforation, and hydrosoluble contrasts should be preferred. Delayed UGI are of great value to evaluate the number and the severity of stenosis when they occur, i.e., since the third week.

Upper endoscopy remains the cornerstone to define the extent and severity of the injury. Even if debated, as the absence of symptoms and signs does not exclude a serious injury, we believe that a panendoscopy should be done by a multidisciplinary team, under general anesthesia, using all available means, in every patient who is suspected of caustic ingestion. Endoscopy should be performed within 24–48 h of the injury before the esophageal wall begins to weaken [4, 21, 22]. The later it is performed, the higher is the risk of perforation. Rigid endoscopes give a better view of the upper airways, the trachea, and the esophageal omentum. The newest small diameter fiberscopes (ø 6 mm) allow for less traumatic exams of the body of the esophagus, down to the stomach including intra-stomachal version.

In any case, the initial endoscopic evaluation must include the larynx and the upper airways, as associated lesions are not unusual: 15 % in our experience. In the de Jong series, 5 % of patients had the hypopharynx primarily involved with no evidence of oral cavity injury. About 12–20 % of patients could have concurrent esophageal injury without any oral pathological finding. Reversely, in spite of some oral lesions, over 70 % of children are free of significant visceral involvement [3, 4, 18, 21, 22].

Practically based, prior to endotracheal intubation, an assessment of laryngotracheal injury is performed with a rigid endoscope. This initial evaluation should include vocal cord movements as paralysis can occur at the time of the caustic injury. Then the child is intubated and the upper esophagus is explored using first the rigid endoscope then the body of the esophagus and the stomach with a flexible fiberscope. If done earlier, the esophagoscopy should reach the stomach, but in case of delayed endoscopy, it is wise to stop at the level of the most proximal circumferential injury. The authors advocating complete esophagoscopy stress the high mortality rate associated with full-thickness necrosis of the lower esophagus and stomach requiring early recognition and intervention. This is true in adults who swallow large amounts of caustic for suicide but very unusual in children. The length of the intact proximal esophagus above the first stenosis should be carefully measured to anticipate swallowing problems. Under view control, a nasogastric tube has to be placed during the initial endoscopy.

Due to stagnation, lesions are more frequent and more serious at the level of anatomic narrowings of the esophagus (cricopharyngeal area, aortic arch left main bronchus and above the esophagogastric junction). Grading of the endoscopic lesions should be helpful to give a prognosis and define the treatment. Unfortunately, there is no common grading system and reported data are not comparable. Our concern is to assess the presence of a lesion, if it is partial or circular and to evaluate its length and depth. The former classification in four grades by Estrera has been implemented by Zargar, describing two subgroups in grade 2 and 3 (a and b), making a difference whether there are ulcers or pseudomembranes [23–25]. As Rossi, we consider that the classification proposed by the Italian Consensus on Not Bleeding Emergency Endoscopy (AIRONE 2008) summarizes most of them and is easily usable [3, 25, 26] (Table 16.1). To summarize, Grade 1 injuries are superficial, Grade 2 are transmucosal, and Grade 3 and 4 refer to transmural injuries. However, precise endoscopic description of the lesions must be very accurate and should be documented with photos and/or videos.

Patients with Grade 0 or 1 are unlikely to have a complicated course or develop complications.

The patients are usually observed for 24 h, fed under supervision, and once tolerated, are discharged.

However, they must be recontrolled on short and long terms. Should any dysphagia or other symptom occur, a UGI should be done to look for a delayed stenosis.

Patients with Grade 2 are treated the same way but more slowly and systematically have a UGI done between 4 and 6 weeks from injury as 50 % of patients of Grade 2b injury may develop strictures requiring dilatations [4].

There is no define treatment for more severe cases and they must be evaluated from case to case. If a NG tube has been placed during the initial endoscopy, it is used to start early enteral nutrition. However, we must consider the placement of a gastrostomy, as the treatment will last long (see below).

Recently, technetium-labeled sucralfate scan, as described by Millar, has been used as a useful and cost-effective screening method to confirm or exclude significant injury, thus avoiding endoscopy [27]. The sucralfate adheres to inflamed mucosa which is recorded on a scan. Patients without any significant adherence should not have a significant injury and could be discharged without follow-up [27, 28]. Computer tomography or magnetic resonance imaging is helpful to assess a perforation and precise its level.

Table 16.1 Classification for caustic injuries in children [25, 26]

Grade	Endoscopic features	Extent of lesions
0	No lesion	
1	Erythema of the mucosa	
2a	Pseudomembranes	Partial/noncircumferential
2b	Ulcer/necrosis	Partial/noncircumferential
3a	Pseudomembranes	Circumferential
3b	Ulcer/necrosis	Circumferential
4	Full-thickness changes/perforations	

16.2.5 Initial Treatment of Caustic Injuries

16.2.5.1 Antibiotics, Corticoids, PPI

In most teams, caustic ingestions are routinely given antibiotics, steroids, and H2 blockers (PPI) [2–4].

Antibiotics seem useless to prevent bacterial colonization of the esophageal lesions as it occurs in a devascularized tissue where microcirculation has been destroyed, so their routine use is debatable. They are indicated in case of perforation and respiratory involvement. Riffat suggests that there is evidence of a lower rate of stricture formation with the use of antibiotics: by decreasing bacterial counts in the necrotic tissue, superinfection is reduced which may lessen the stricture formation [2]. Occasionally, we have observed a peak of fever after dilatation in some children. As cerebral abscesses have been reported in such circumstances, those cases received a prophylactic dose of antibiotics before each dilatation without recurrence of fever [29].

The beneficial role of steroid on inflammation and scarring process is still debated. They could be used in first- and second-degree injuries but not in third degrees because of the potential increased risk of perforation. Some cases are reported who developed a gastric ulcer with associated hemorrhage after receiving systemic steroids [18]. A meta-analysis of 13 studies done by Fulton over 50 years has concluded that steroid use does not decrease the incidence of stricture formation following Grade 2 caustic ingestion, and therefore, the use of steroids was not advised [30]. Some reports, more specifically concerning infants and toddlers, have shown that the use of high dosage of corticosteroids, starting at the early phase of treatment, could be beneficial in decreasing the need for dilatations [31–33]. Our experience is that corticoids do not prevent from esophageal stenosis in serious caustic burns, but are helpful to achieve faster resolution of the edema, mainly on the airways.

The caustic burn induces a shortening of the esophagus and a motility disorder resulting in reflux with poor esophageal clearance, which adds a peptic stenosis to a caustic one as evidenced by histology (O. Reinberg, unpublished). For this reason, as many others, all our patients under conservative treatment with dilatations receive proton pump inhibitors (PPI) even if their efficiency has not been proven [2–4, 18].

16.2.5.2 Gastrostomy

Benign esophageal strictures usually produce dysphagia for solids, liquids, or both, with slow and insidious progression of weight loss and malnutrition. If the stenosis is important with subsequent dysphagia lasting for more than a month, a gastrostomy should be done to avoid long-lasting total parenteral nutrition with its potential complications. Most patients referred to us, even those with a previously done gastrostomy, were in poor nutritional conditions and must be placed under refeeding program before surgery.

Percutaneous endoscopic gastrostomy (PEG) is our favorite technique for feeding tube placement in children with inadequate nutritional intake. However, it is not feasible after caustic ingestion and a gastrostomy has to be performed. As most of our patients are in poor conditions, they require a gastrostomy including a gastropexy to avoid parietal disunion related to

malnutrition. We have described a technique of a real Stamm gastrostomy performed by laparoscopy for these cases [34]. This laparoscopic technique combines the advantages of a minimal invasive procedure with the safety of an open operation and related gastric attachments to the abdominal wall.

The proper placement of the gastrostomy on the anterior stomach wall is a major concern. When intending to replace an esophagus, the surgeon never knows which transplant can be used: if the gastrostomy has been placed too close from the greater curvature, he may face an interruption of the gastroepiploic artery and a gastric tube cannot be achieved. When performing a gastrostomy for caustic stenosis, it is wise to place it far away from the great curvature, just in case a tube could be done.

Gastrostomies are our first choices of surgical access to the bowel, better than jejunostomies. However, if the stomach has been involved in the caustic injury, we must refrain from using it and then perform a jejunostomy.

In some cases, we used an interesting artifice suggested in 1974 by Papahagi and Popovici: when performing the gastrostomy, these authors ligated the middle colonic artery and sometimes the right one to stimulate the development of the left one, anticipating a transverse isoperistaltic colonic replacement [35].

16.2.5.3 Dilatations

About a month after caustic ingestion, once the edema has gone, the diagnosis of stenosis can be assessed by an esophagogram and an endoscopy. Then, according to the severity of the stenosis, a dilatation program can be started. The rate of stricture formation reported in literature varies from 2 to 63% (!). Isolated short stenosis of the esophagus, i.e., 1–2 cm, can be treated by dilatations with good results. Long ones (more than 3 cm), multiple stenosis (more than two), or those with a tracheoesophageal fistula cannot be solved by dilatations and require an esophageal replacement [36, 37]. However, the decision should not be precipitated.

Of the various methods to dilate, we use three of them: the Tucker-Rehbein bougie on a never-ending loop, the Savary-Gillard bougie on an atraumatic guidewire (M. Savary was our Chief of ENT in Lausanne, Switzerland), or the balloon dilators similar to angioplasty. The Tucker-Rehbein bougie has the advantage of being done without endoscopy or chest X-rays control, with a very low risk of perforation. It requires placing a string from the nose, down into the esophageal lumen, and externalized through the gastrostomy. It can be used both ways, antegrade or retrograde. To dilate, the Tucker-Rehbein bougie is tied to either ends of the string and pushed or pulled using progressively larger dilators. When using balloon dilatators, a radial pressure on the stricture is performed that is thought to be better than a longitudinal direction of dilatations as done with the other methods. But balloon dilatation is not as safe as described, as it can be difficult to control the strength of expansion when the balloon inflates suddenly. For this reason the Savary or the Tucker-Rehbein's techniques are softer and more progressive. Our belief is that all different techniques should be available in a team caring with caustic burns and be adapted to each case and dilatation.

The optimal frequency of dilatation is not well established in the literature, and our practice was to use a symptom-based approach, but an interval of 3 weeks seems appropriate in most cases. We encourage normal eating as soon as possible, as pieces are good self-dilatators, but with a high risk of entrapment. The scarring process of the esophagus is long, and the evolution of a stenosis must be confirmed by repeated esophagograms. An important apparent stenosis related to the inflammatory process can last for months before its disappearance. On the other hand, a dilatation program without significant improvement after a year can be considered as a failure. For these reasons we do not continue a dilatation program more than 1 year. However, some teams persist in dilating patients for years, up to 15 years [18]. Dilatation should be continued as long as a progressive increase in esophageal diameter is noted, along with the recovery of a normal feeding. Even after, dilatation has to be continued from time to time. Without improvement at 12 months, we consider doing an esophageal replacement. Indications for

esophageal replacements and their timing vary widely. As a result, children are often subjected to prolonged courses of dilatations prior to esophageal replacement or, conversely, may be exposed to unnecessary surgery [36]. A strong predictor of poor outcome was the delay from ingestion to beginning of dilatations [36, 38].

16.2.5.4 Stents

The early insertion of stents was first proposed by Salzer in 1920 and later advocated by Fell [39, 40]. Early reports fell into disfavor because the strictures soon reformed after removal of the stent [18]. However, Coln as well as Estrera wrote that after stenting, the frequency of recurrences decreased and the strictures were easier to dilate [23, 41]. The use of a self-expanding esophageal stent for malignant strictures is well documented first on animals and now applied in human cases of malignant and recalcitrant benign strictures [42–45]. It has evolved toward early endoscopic esophageal stenting using removable plastic or metallic self-expandable stent [46], or better using biodegradable stents [47, 48]. These techniques are under evaluation and the new materials available are promising. They could play an interesting role to prevent stenosis; however, many migrations or displacements are described [3, 46]. Recently, Okata published the histology of a removed esophagus after self-expandable biodegradable stenting and was able to compare the histology of the esophageal wall under the stent and at distance from its ends. The resected specimens showed thickened scar formation at the level of the stricture, while the degree of esophageal wall damage, both at the proximal and distal ends of the stricture, was slight [48, 49].

The idea is that the stent prevents the adhesion of the facing surfaces of the esophagus, thus minimizing the stenosis, but they cannot restore the defect of the muscular layers. Even if the lumen remains open, a rigid, nonpropulsive segment of the esophagus will be left. Until now we refused to use them as we have been referred 11 children with major complications after esophageal stenting: migrations in the mediastinum and in the left bronchus and posterior erosion of the trachea; one of them 7 years old having had 42 previous procedures.

16.2.5.5 Other Treatments

Many agents have been tried as adjuvant therapies in order to prevent excessive granulation tissue formation.

Mitomycin C is an antibiotic-cytostatic drug derived from *Streptomyces caespitosus* similar to antialkylating agents. It inhibits DNA and protein synthesis by inducing cross-linking, thus fibroblast proliferation. It is used in multidrug regimen in oncology for disseminated carcinoma as well as for transitional cell tumor of genitourinary tract, but it has a poor antimitotic effect. However, its properties have led to its use as an agent for reducing scar formation in ophthalmology for the treatment of pterygium surgery since 1963 and of refractory glaucoma since 1983, and it is commonly used today in those fields even in children. It has been used by ENT surgeons for recurrent laryngeal or tracheal stenosis both in adult and children. We first presented the use of mitomycin C in recurrent esophageal strictures in children in 2001 at the 33rd annual meeting of the Canadian Association of Paediatric Surgeons [50], followed by Afzal who published the first pediatric cases [51]. The use of mitomycin C is still limited in this indication with some cases or very small series reported [52–54]. In our team, first we dilate the stenosis with Tucker-Rehbein or Savary bougies, then the mitomycin C is applied by the ENT surgeons through a rigid esophagoscope. Two ml of mitomycin C Kyowa® solution 2 mg/ml are applied for 2 min using a peanut positioned on the area uncovered with mucosa under visual control. We have the experience of 25 pediatric cases treated with 1 to 4 topical application of mitomycin C after dilatation either for recurrent esophageal stenosis or for stenosis of the upper anastomosis after esophageal replacement with a success rate of 82 %. Other authors came to the same conclusions (El-Asmar 2015, 21 children, 86 % success [55]).

Hyaluronic acid is used in many clinical situations as diverse as neurosurgery and wound healing. A study showed that hyaluronic acid treatment could be effective in treating damage

and preventing strictures after experimental caustic esophageal burn on rats [56]. Several other different chemical agents (heparin, vitamin E, caffeic acid phenethyl ester, tamoxifen, 5-fluorouracil) have also been used experimentally, but only a few of these have been added to clinical treatments. Most of these agents impair collagen metabolism and inhibit fibroblastic proliferation either by direct or indirect routes. Physical treatments have also been used such as argon plasma coagulation, but they remain anecdotal [57].

Surgical segmental resections followed by end-to-end anastomosis have a very high rate of failure even after adding enlargement procedures. Unlike the resection of a congenital esophageal stenosis where the anastomosis is performed in normal tissue on both sides of the malformation, the resection of a caustic stenosis is always done in an injured pathologic tissue and leads to recurrence of the stenosis as done under tension in a poorly vascularized tissue.

16.3 Foreign Bodies

16.3.1 Ingested Foreign Bodies (FBs)

The ingestion of foreign bodies (FBs) is a common problem in infants, but fortunately the majority of them will pass through the digestive tract without any adverse effects. The peak incidence of FB ingestion is between 6 months and 3 years, and coins are the most common with an occurrence of >125,000 ingestions per year (2007) and 20 deaths reported in the United States during a 10-year period [58–60]. It has even been described in neonates (esophageal zipper in a 2 months old baby) [61]. Some kids continue to put unbelievable objects in their mouths after infancy. Coins, toys, crayons, and ballpoint pen caps are most often ingested during the childhood [62, 63]. Food impactions are not as frequent as in adults but not unusual [63].

The dangerous FBs are those who remain entrapped in the esophagus at the level of anatomic narrowings: esophageal omentum, aortic arch and left major bronchus, and above the esophagogastric junction. There are no guidelines available to determine which type of object will pass safely. The size depends on the age of the child. A study done by Tander on 62 ingestions in children tries to correlate the sizes of the FB with the ages: up to 5 years of age entrapments occurred for objects between 17 and 23 mm, and after 5, objects from 23 to 26 mm were involved [64].

A FB impacted in the esophagus leads to a pressure lesion and local necrosis resulting in stenosis or perforation. Once in the stomach, it may pass through the pylorus and be eliminated in the stools. But it can be retained anywhere along the bowel at places of anatomical narrowing or angulation such as duodenojejunal flexure or ileocecal valve causing mechanical obstruction. If the object has irregular or sharp edges, it may lodge anywhere in the GI tract. If the objects are elongated, they can become trapped in the appendix or ileocecal valve.

16.3.2 Management of FBs

After ingestion, children can be asymptomatic at the time of presentation. If present, common symptoms include drooling, gagging, dysphagia, odynophagia, decreased appetite, food refusal, neck pain, chest pain, abdominal pain, cough, stridor, wheezing, and respiratory distress. Esophageal FBs often present with respiratory complaints. In most patients physical examination is normal [59, 63].

The priority is to assess the presence of a FB.

Chest radiographs including the neck, and a supine abdominal one, should be obtained to rule out ingestion. Two orthogonal projections are mandatory, because some FBs, especially those of discoid shape, could be shown only in one view [59, 62]. Limited chest radiograph not including the upper thoracic inlet may miss a higher-up foreign body. Radiological visualization depends on radiopacity. Radiograph detects as much as 80 % of all FBs [62]. Objects of metal, except aluminum, most animal bones, and glass are opaque on radiographs. Objects composed of plastic and most fish bones are radiolucent structures, and their diagnosis may be challenging.

Careful attention should be placed on the edges of a presumed coin to exclude the double halo typical of a button battery, which may easily be mistaken for a coin. Regarding FBs, such as fish bones, chicken bones, and toothpicks, an X-ray has a sensitivity that ranges from 23 to 55 % for the first two and 9 % for the latter. In case of toothpicks, even other imaging studies have a low sensitivity, 15 % for MDCT and 29 % for ultrasound (US). But they are the methods of choice in the diagnosis of a FB that migrated from the GI tract and retained in the soft tissues [62, 65].

An expert panel from the North American Society for Pediatric Gastroenterology Hepatology and Nutrition (NASPGHAN) was convened and produced the following guidelines for practical clinical approaches to the pediatric patient with a variety of FB ingestions [63].

Symptomatic FBs impacted in the esophagus have to be removed urgently. Asymptomatic FBs in the esophagus should be removed within 24 h to reduce the risk of significant esophageal injury or erosion into neighboring structures.

Once in the stomach, FBs can generally be managed expectantly in asymptomatic patients. Parents should be instructed to monitor the stools for passage of the FBs. X-rays should be obtained every 1–2 weeks until clearance can be documented. If the FB is still retained in the stomach after 2–4 weeks of observation, elective endoscopic removal may be considered. Children with underlying anatomic or surgical changes, such as previous pyloromyotomy, have an increased risk for retained FBs [63, 66, 67]. Just before removal of a retained gastric FB, X-rays should be repeated to make sure that the FB has not passed just before.

The gold standard is endoscopic removal under general anesthesia. Most ingested FBs are best treated with flexible endoscopes, which allow retrograde exploration of the gastric fundus. However, rigid esophagoscopy may be helpful for proximal foreign bodies impacted at the level of the upper esophageal sphincter or hypopharyngeal region. Various retrieval devices are used, including rat-tooth and alligator forceps, polypectomy snares, polyp graspers, Dormier baskets, retrieval nets, magnetic probes, etc. Before endoscopy, practicing grasping test on an object similar to the ingested one may help to determine the most appropriate available retrieval device and in what fashion the object has to be seized.

However, nonendoscopic methods have been successfully used. The very high esophageal FBs can be retrieved with forceps under direct laryngoscopy. We have a good experience with the use of a Foley catheter under fluoroscopic guidance to "sweep" out coins lodged in the esophagus, while the patient is maintained in the prone Trendelenburg position [68–71]. It can be done without anesthesia in selected patients. The positioning of the Foley catheter can be helped with some contrast in the balloon. Esophageal bougienage is another technique that uses a blunt Hurst dilator to push down an esophageal FB into the patient's stomach [72–74]. It is safe and cost-effective compared with endoscopic removal. The disadvantage is that direct inspection of the esophagus for underlying pathology is not done as well as inability to retrieve the FB, which "falls" in the stomach.

16.3.3 Particular Cases

16.3.3.1 Button Batteries

Button batteries represent a special category of pediatric ingested foreign body because of their potential for severe morbidity and mortality particularly if impacted in the esophagus [75–81]. The number has increased by 80 % between 1998 and 2008 due to their large use in toys and electronic devices [81–83]. Ingested button batteries have been reported to cause esophageal stricture and perforation [75, 76, 79], vocal cord paralysis [80], tracheoesophageal fistulas, and even deaths [63, 78, 81].

Button battery cells generally contain a heavy metal like mercury, manganese, silver, and lithium and a strong hydroxide of sodium or potassium. The quality of the sealing between anode and cathode is highly variable. Some of them can resist hours in gastric acid. Reversely, others are quickly dissolved with a leak of the potentially toxic or corrosive content leading to intoxication

or mucosal damage by ulceration, which may further lead on to perforation and secondary stricture formation [80]. The damage can also be due to electrical discharge leading to low-voltage burns, and pressure necrosis especially in the esophagus [81]. These very severe complications can occur within a few hours as experienced on animals and be proven in toddlers (transmural necrosis of the esophagus within 3 h) [81, 84].

Once in the stomach, batteries rarely cause any harm to the gastric wall, and its spontaneous passage through the pylorus is expected. Subsequently, conservative management is a generally accepted alternative. However, gastric perforation has already been described 2 days after ingestion [85]. Even after conservative management, these patients have to be followed up as distal bowel disturbances have been described such as Meckel's diverticulum perforation or impaction in the ileocecal valve [86].

Consequently, overall consensus is that batteries lodged in the esophagus should be removed immediately. Opinions differ on the management of those located in the stomach in children. We follow the protocol suggested by Eisen [87], waiting to see whether the battery will spontaneously pass through the pylorus within 24 h, as long as the patient manifests no sign of injury to the stomach. When batteries are retained longer in the stomach and/or the patient becomes symptomatic, we attempt endoscopic removal.

16.3.3.2 Magnets

Pediatric magnet ingestions have received increasing attention over the past 10 years. Although most of those small smooth ingested FBs will pass spontaneously through the gastrointestinal tract, multiple magnets are a danger of being able to attract each other through different loops of bowel, arresting their movement, and causing transmural pressure necrosis. This can lead to bowel perforation, fistula formation, volvulus, obstruction, intra-abdominal sepsis, and death [88–90].

The US Centers for Disease Control and Prevention has reported one death and 20 surgeries for bowel perforations related to magnet ingestions between 2002 and 2006 [91]. In 2011 there were 30 publications on such cases with more than 100 bowel perforations in children. The US National Electronic Injury Surveillance System showed that the rate of magnet-related injury had increased dramatically over the period from 2002 to 2011 [90, 92], as did the Consumers' Federation of Australia [92], the Hospital for Sick Children in Toronto, Canada [88], and the Surgical Section of the American Academy of Pediatrics [93]. These changes are related to the documented technological shift from ferrite magnets to neodymium-iron-boron magnets that are approximately 10–20 times more powerful. They are often sold as sets of multiple spheres approximately 5 mm in diameter or as parts of toy construction kits. In 2013, Health Canada issued a recall of neodymium-iron-boron magnet sets marketed as desk toys [94]. The United States Consumer Product Safety Commission established a mandatory standard to prevent magnets detaching from toys. This standard also prohibits magnets and loose magnet components in toys for children under age 14 years [95]. Unfortunately, hundreds of thousands of magnet sets have already been sold, and despite these regulations, vendors via the Internet continue to sell these products. The North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition released survey results in 2012 demonstrating that despite increasing warning labels, these labels were ineffective at preventing ingestion [96].

The median age at ingestion is between 2 and 5 years of age. But parents and caretakers should counsel teenagers and young adults of the hazards of fake body piercings that use small magnet backs. Consumer Product Safety Commission reported instances of teenagers swallowing magnets unintentionally when placed on the opposite sides of tongue jewelry to mimic body piercings [97].

Most patients are asymptomatic. A plain abdominal radiograph is recommended at admission if magnet ingestion is suspected as they are radiopaque FBs. However, X-rays and computed tomography lack the sensitivity to determine the number of magnetic objects, thus making management decision difficult. Some authors consider that any ingestion should be treated as though multiple magnets were ingested [98].

If case of ingestion of an attested single magnet and if its size is small enough to pass spontaneously, the child could be managed by observation only. If multiple magnets are ingested or if their actual number cannot be determined as it occurs in most cases, intervention is required. If the magnetic FB remains in the esophagus or in the stomach, it should be removed by endoscopy or using a magnetic probe. Once multiple magnets have passed the pylorus and remain in the duodenum, an attempt of endoscopic removal could be done. In case of failure to remove multiple magnets endoscopically or if they are already in the jejunum or below, surgical intervention is required to avoid further complications. This can be done by laparoscopy with umbilical extraction or by laparotomy [93, 98–100].

16.3.3.3 Sharp and/or Long Objects

Ingestion of large and/or long objects is also an issue of special concern. The reported incidence of FBs causing perforation of the GI tract is less than 1 %, with the objects being elongated or sharp in most of the cases, such as toothpicks, pins, fish, or chicken bones [59]. Furthermore, long, narrow, and pointed ingested FBs >5 cm in length (3 cm in young children) are unlikely to clear the duodenal sweep and, if they do, are equally unlikely to pass through the ileocecal valve [59, 63]. Subsequently, large or long objects, even though they are blunt, should be removed from the stomach. Given the low risk of endoscopy and albeit rare but significant risk of severe morbidity and mortality from swallowed sharp objects, removal of all of them within the reach of the endoscope is recommended [63].

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