Esophageal Injuries

Денис Овечкін

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Injuries to the esophagus in children are often due to accidental ingestions or traumatic injuries as young children have a tendency to explore the world with their hands and mouth.

Ingestion of various foreign bodies including peanuts, phytobezoars, trichobezoars, and pica in the mentally disabled can cause partial or complete bowel obstruction in children.

The majority of foreign body ingestions occur in children between the ages of 6 months and 3 years. Coins are the most common foreign bodies ingested by children. Other objects, including toys, toy parts, magnets, batteries, safety pins, screws, marbles, bones, and food boluses have been reported.

The size, shape and characteristics of the object dictate initial management: either continued observation or efforts at immediate retrieval.

Most complications from pediatric foreign body ingestion are due to esophageal impaction, usually at one of the 3 physiological narrowing areas: 1) at the upper esophageal sphincter or thoracic inlet (70%); 2) in the mid-esophagus at the level of the aortic notch (15%); 3) just above lower esophageal sphincter (15%).

Once the foreign body has reached the stomach in a child with a normal GI tract, it is less likely to lead to complications.

Problems occur at fixed points or sites of anatomical narrowing or angulation such as the pylorus, the C-loop of the duodenum, the ligament of Treitz and the ileocecal valve.

Fortunately, most foreign bodies that reach the gastrointestinal tract pass spontaneously (usually within 24 to 48 hours). Only 10-20% will require endoscopic removal, and less than 1% require surgical intervention.

Battery ingestion poses a special problem. Because batteries may leak or burst within 24 hours of ingestion and cause corrosive alkali burns or poisoning from mercuric salts, they are removed endoscopically while still in the stomach. Once they have passed into the duodenum, they are removed surgically if they do not progress rapidly through the intestine as indicated by roentgenograms taken at 6-hour intervals.
Clinical Presentation

Children may complain of dysphagia, develop sialorrhea, or may develop respiratory symptoms from compression of the membranous trachea. Less frequent complaints include odynophagia and chest pain. The presence of cervical swelling, erythema or crepitus raises concern for an oropharyngeal or proximal esophageal perforation.

Diagnosis

- Posterior and lateral neck radiographs and a chest X-ray are needed to identify the location of the object and evaluate for extravasated air in the mediastinum, subcutaneous tissue, or chest.
- Water soluble contrast may be used to identify the object when plain films are negative and clinical suspicion is high.

Complications

Complications of esophageal foreign bodies include perforation, aspiration, retropharyngeal abscess, mediastinitis, pericarditis, pneumothorax, pneumomediastinum and vascular injury.

Treatment

Patients need no hospitalization but should return if they develop abdominal pain, vomiting, bloody stools, or if the object has not been identified in the stools in 4-5 days. The patient's stools are strained for the object and tested for blood. Roentgenograms document the progress of the object or whether it has passed unnoticed. Surgical removal is indicated for continued abdominal pain, vomiting, significant bleeding, or failure of the object to pass in 4-5 weeks [14].

Esophagus:

Esophageal foreign body impactions should be investigated and removed as soon as possible to prevent aspiration and perforation [2, 3, 14, 43]. Urgent endoscopic intervention is required an object is located in the upper third of the esophagus. If the object lies in the middle to lower esophagus, a repeat film should be obtained (prior to endoscopy as the object might have migrated) and endoscopy performed within 12-24 h if the child is asymptomatic.

Stomach:

Foreign bodies in the stomach are likely to be eliminated within 30 days [14, 43]. Most foreign bodies that reach the stomach will pass easily. If it has not passed in the feces in 4-6 weeks, endoscopy is recommended for removal.
Coins that reach the stomach, depending on their size and patient's age, are likely to pass without complication and can usually be simply followed radiographically.

Button batteries, sharp objects, and objects causing bleeding, acute or severe airway compromise, or significant pain or dysphagia require emergent removal.

Children should resume a regular diet and the stool should be strained for passage of the foreign body.

**Intestine and colon:**

If an object is beyond the stomach but remains in the same location for more than 1 week, surgical removal should be considered [14, 36, 43]. Fever, vomiting, abdominal pain or peritoneal signs are indications for immediate surgical removal.

**Urgent intervention is indicated (common recommendation):**

- When the ingested object is sharp, long (>5 cm), consists of multiple magnets, and is in the esophagus or stomach.
- When a disk battery is in the esophagus (and in some cases in the stomach)
- When the patient shows signs of airway compromise.
- When there is evidence of near-complete esophageal obstruction
- When there are signs or symptoms suggesting inflammation or intestinal obstruction

**Approaches for specific types of foreign bodies (common recommendation):**

**Coins:** usually can be observed for up to 24 hours after ingestion as 20-30% will pass into stomach spontaneously. Esophageal coins should be removed promptly if patient is symptomatic or does not pass esophageous spontaneously by 24 hours after ingestion.

**Disk batteries:** a medical emergency, as contact with esophageal wall with both poles of battery can conduct electricity which can rapidly lead to necrosis and perforation of esophagus. However, once passed to stomach, most pass harmlessly.

**Sharp pointed objects:** if lodged in esophagus, represents a medical emergency because 15-35% risk of perforation. Endoscopy should be performed because many sharp pointed objects are not readily visible by x-ray.

**Magnets:** a single ingested magnet is usually low risk, however two or more magnets may attract across layers of bowel leading to necrosis, fistula, perforation or obstruction. Thus, location, and number of ingested magnets are important to determine and multiple magnets ingestion indicates preemptive removal.

**Outcomes**

Perforation and bleeding are the most frequent complications of extraction and occur in 2-13% of cases [14].
**CAUSTIC ESOPHAGEAL INJURY**

Caustic esophageal injuries occur most frequently in male children under age 3.

**Etiology**

The type and extent of injury resulting from ingestion of a caustic solution depends upon:

1. the type of agent
2. its physical state (i.e., liquid, solid)
3. concentration
4. the amount ingested
5. the duration of contact with the esophageal or gastric mucosa

The most frequently ingested caustic agents are alkaline agents. Most cause only limited injury to the esophageal mucosa without extensive necrosis or subsequent sequelae.

The most common sources of alkaline ingestion causing caustic injury include: household lyes (potassium and sodium hydroxide), drain cleaners (sodium hydroxide), dishwashing detergent and household cleaners (sodium metasilicate and ammonia).

Strong acids frequently have a bitter taste, emit a strong odor and burn on contact, which often results in rapid expulsion after ingestion. When swallowed, acids usually cause significant damage to the stomach and variable mucosal damage to the esophagus. The duodenum and proximal small bowel are relatively well-protected by the pylorus.

**Pathophysiology**

Injury to the mucosal surfaces occurs within seconds of the insult. The ingestion of corrosive agents can cause devastating injury to the esophagus and stomach.

Liquid forms cause more significant injuries than solid products. Household bleach, liquid laundry detergents, and ammonia usually result in mild esophageal burns since they are weak bases at low concentrations. Ammonia, also results in chemical pneumonitis and pulmonary edema. Ingestions of strong alkalis such as liquid lye, dishwasher cleaner, oven or toilet bowel cleaner, or button batteries are associated with the most deaths since they represent the highest concentration of a strong base.

The physical form and pH of the corrosive agent play a significant role in the resultant injuries. Substances with pH values less than 1.5 or greater than 12 result in the most severe injuries [14].

The nature of the injury differs between acid and alkali ingestion. Alkalis produce a liquefactive necrosis with rapid tissue penetration. Alkali ingestion causes destruction of the epithelium and submucosa with occasional extension into the muscularis. A friable eschar is formed and neutralization follows; however, continued destruction of the deeper layers is still possible following alkali ingestion.

Acids cause a coagulation necrosis and a superficial hard eschar. This coagulum limits the extension of further mucosal penetration beyond the surface burn.

Caustic injuries to the esophagus are classified similarly to thermal burn injuries of the skin (Table 2.4). Table 2.4.

**Classification of caustic injuries**

<table>
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<th>Grade</th>
<th>Depth of Mucosal Involvement</th>
<th>Endoscopic characteristics</th>
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<tr>
<td>0</td>
<td>None</td>
<td>Normal esophagus</td>
</tr>
<tr>
<td>I</td>
<td>Superficial mucosal burn</td>
<td>Mucosal edema and hyperemia</td>
</tr>
<tr>
<td>II A</td>
<td>Transmucosal injury</td>
<td>Noncircumferential, patchy, superficial ulcerations, exudates, mucosal sloughing over &lt;1/3 esophageal length</td>
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<tr>
<td>II B</td>
<td></td>
<td>Circumferential injury, deeper ulcerations, exudates, mucosal sloughing over &gt;1/3 esophageal length</td>
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Caustic ingestion injury is described in three phases:

- the acute phase (0-4 days),
- the subacute phase (5-14 days),
- the cicatrisation phase.

The acute phase is characterized by the presence of inflammation, edema, thrombosis, eschar formation and necrosis. The subacute phase or reparative phase occurs between 5 and 14 days following the injury. During the subacute phase, necrotic tissue is sloughed, fibroblasts are deposited and neovascularization begins. The esophageal wall is weakest during the subacute phase and at this time is most prone to perforation. The cicatrisation phase occurs between 3 and 6 weeks following injury.

Fibrous tissue replaces the submucosa and muscularis forming dense scar that results in strictures, obliteration, or shortening of the esophagus (Fig. 2.1).

**Clinical Presentation**

The clinical presentation of children following caustic ingestion is highly variable. Signs and symptoms include drooling, burning pain in the mouth and lips, odonyphagia, dysphagia, hoarseness, stridor and aphasia.

Fever, tachycardia and hypotension are signs of severe esophageal injury and often indicate massive ingestions.

Esophageal perforation is often associated with symptoms of severe retrosternal, back, or upper abdominal pain.

Physical signs of caustic ingestion include ulceration or discoloration of the oropharyngeal mucosa, cervical crepitance, hematemesis and peritonitis.

**NB:** A lack of external physical findings does not exclude the possibility of caustic ingestion.

**Diagnosis**

*Fiberoptic endoscopy* is both accurate and safe, especially when done *within 24 to 48 hours after ingestion.*

Unnecessary treatment is avoided when esophageal injury can be excluded.
NB: Because the history and physical examination are unreliable in assessing the degree of esophageal involvement, endoscopic examination of the oropharynx and upper gastrointestinal tract is crucial.

In severe cases the plain chest and abdominal X-ray examinations are indicated to evaluate for mediastinal or free intraperitoneal air indicating perforation. If fever, systemic sepsis, and upper abdominal signs are present, perforation may have occurred and a water-soluble contrast esophagram may be useful to provide evidence of perforation. A contrast esophagogram is usually done after 10 to 14 days, when an assessment of the entire esophagus and upper gastrointestinal tract can identify the extent of injury and may help in choosing the appropriate therapy.

Technetium-labeled sucralfate radioisotope scanning of the esophagus has been used successfully as a screening device, with lack of sucralfate adherence indicating the absence of significant injury [43].

Intensive care unit observation is necessary to monitor for signs of worsening or more complicated injury (i.e., esophageal perforation, gastric perforation, tracheoesophageal fistula, mediastinitis).

Treatment
For patients with first-degree burns (grade I injury), no specific treatment is necessary. Liquid oral intake is initiated and extended to solids. If solid foods are tolerated, the child can be discharged. Clinical follow-up at 2 to 3 weeks is indicated, and contrast examination is done if residual clinical symptoms of dysphagia are noted. Our management protocol is summarized in Addition C (See Additions).

Patients with moderate (grade IIa) or severe (grade IIb and III) injuries require further treatment aimed at the prevention of stricture formation because up to 50% will develop strictures [43]. Although most patients with grade IIa injuries recover completely, close follow-up is required and endoscopy and dilatation must be done as prophylactic measures [7, 8, 43].

Major controversy surrounds the treatment options for severe injuries - namely, the use of steroids and antibiotics, esophageal stents, and esophageal dilatation.

Grade IIIb injuries are rare in the pediatric age group and usually occur in adolescents attempting suicide. These injuries may require immediate and aggressive surgery if extensive necrosis and perforation are present, especially if the stomach is also involved.

Prehospital Care and Initial Management
Attempt to identify the specific product, concentration of active ingredients, and estimated volume and amount ingested. The product container or labels may be available.

Gastric emptying and decontamination
Do not induce emesis or attempt to neutralize the substance by using a weak acid or base. This induces an exothermic reaction, which can compound the chemical injury with a thermal injury. It may also induce emesis re-exposing tissue to the caustic agent. Do not administer emetics because of risks of re-exposure of the vulnerable mucosa to the caustic agent. This may result in further injury or perforation. Small amounts of a diluent, although controversial, may be beneficial if administered as soon as possible after a solid or granular alkaline ingestion, to remove any adhering particles to the oral or esophageal mucosa. Water or milk may be administered in small amounts. It is very unlikely to be of any benefit after more than 30 minutes [14].

Gastric lavage by traditional methods using large-bore orogastric Ewald tubes are contraindicated in both acidic and alkaline ingestions because of risk of esophageal perforation and tracheal aspiration of stomach contents. Large-volume liquid ingestions may benefit from nasogastric tube (NGT) suction if performed rapidly after ingestion. Pyloric sphincter spasm may prolong contact time of the agent to the gastric mucosa for up to 90 minutes. NGT suction may prevent small intestine exposure. NGT suction may be of particular value following ingestion of zinc chloride, mercuric chloride, or hydrogen fluoride, unless signs of perforation are present.

Neutralization
Do not administer a weak acid in alkaline ingestions or a weak alkaline agent in acid ingestions. There is a risk of heat production resulting from this exothermic reaction. In addition, the risk of emesis makes this a hazardous intervention [16, 43].

DO NOT DO 4-things:
Hospital Care

Emergency management
- Close attention to the ABCs (airway, breathing, and circulation)
  - Stridor or aphonia indicate laryngoepiglottic injury and may require urgent orotracheal intubation for airway protection.
  - Occasionally, severe laryngeal destruction necessitates emergency cricothyroidotomy or tracheostomy.
- Adequate vascular access to allow for correction of hypovolemia or hypotension
  - Nasogastric tube placement is not recommended routinely because it may be associated with subsequent stricture formation.
- Initial orders include:
  - Nothing by mouth
  - Proper fluid resuscitation
  - Nutritional support through total parenteral nutrition
  - Pain management
- Antimicrobial therapy - after disruption of the mucosal barrier by caustic ingestion, bacterial translocation and secondary bacterial invasion are likely.
- Nutrition:
  - Parenteral nutrition for patients with grade II and III injuries
  - Oral feedings are withheld until dysphagia of initial phase has regressed. In most cases, oral feeding commences as soon as the patient can swallow saliva.
  - Proton pump inhibitors should be prescribed. Loss of lower esophageal sphincter tone occurs secondary to corrosive esophageal injury. Acid reflux can exacerbate underlying injury and accelerate stricture formation.

The use of steroids (i.e., prednisolone, dexamethasone) and antibiotics in initial management of caustic esophageal injuries is controversial. Steroids inhibit the inflammatory process and may reduce granulation and stricture formation. Unfortunately, steroids are also immunosuppressive and may contribute to infectious complications and morbidity [14, 42, 43]. Although more recently, the use of high-dose steroids (dexamethasone 1 mg/kg/day) has been advocated [43].

Therapy depends on the grade assigned at endoscopy:
Once the grade of injury is determined (Table 2.4), the suggested management is as follows:
- **Grade I**: Children with grade I injuries are admitted for observation and intravenous fluid administration.
  - A clear liquid diet is started at 36-48 hours and advanced as tolerated to a general diet.
  - A contrast esophagram is performed 2-3 weeks following injury in any child with residual symptoms or dysphagia.
- **Grade II/III**: Children with grade II or III injuries are denied oral intake for several days (sometimes weeks). Parenteral nutrition, either peripheral or central, is mandatory to provide adequate nutritional support.
  - The use of high-dose steroids and antibiotics is preferably [2, 3, 6, 8].
  - Oral intake is withheld until patients can tolerate swallowing their saliva. A liquid diet is started initially and advanced to a general diet as tolerated.
  - Severe grade III injuries with esophageal perforation mandate surgical intervention.
  - A barium esophagram is performed at 2-4 weeks following injury to identify early stricture formation.
**Immediate Surgery**

Immediate surgical intervention is indicated in those patients with uncontrollable hemorrhage or perforation (i.e., mediastinal air, intraperitoneal air, or peritonitis).

Esophageal resection can be performed via either thoracotomy or laparotomy (i.e., transhiatal). After resection, a gastrostomy or jejunostomy tube is placed and the proximal esophagus is diverted as a cervical esophagostomy.

Reconstruction of the alimentary tract is delayed for at least 2-3 months or until all acute problems are resolved.

*Surgical options include:*

- Bypass with placement of an esophageal substitute
- Resection
- Both bypass and resection
- Esophagoplasty

**Outcomes**

Long-term complications of corrosive ingestions include:

- Stricture formation of the esophagus
- Gastric outlet obstruction
- Esophageal carcinoma

The most common complication of caustic ingestion is stricture formation of the esophagus (Grade I – rare; Grade II – 20-30%; Grade III – 90-95%) [14, 43].

The incidence of esophageal carcinoma in patients after caustic ingestion is estimated to be 500-1,000 times greater than the incidence in the general population. The latency period between initial injury and development of esophageal carcinoma varies from 10-50 years [14]. Lifelong follow-up and screening endoscopy are necessary.

Once a esophageal stricture has developed, dilatation becomes necessary and is usually started at 6-8 weeks after injury [42].

**NB:** The risk of esophageal perforation with dilatation is relatively low; however, this remains the most common complication. The most experts advocate waiting 6 weeks before initiating dilation, because risk of perforation is highest in the first weeks after injury.

**Esophageal dilation**

Weekly dilatation is continued until the stricture softens.

*Dilation can be accomplished by 2 methods:*

- Pulsion dilation (*Bouginage*) – graded bougies are passed over endoscopically or radiographically placed guide wires. This technique is difficult with tortuous or complicated strictures and warrants fluoroscopic guidance.
- Radial dilation (*Balloon dilation*) – uses endoscopically or radiographically controlled balloon dilation

**Bouginage**

The traditional method of dilating an esophageal stricture is by bouginage.

A variety of dilators are available (mercuryloaded tip, gum elastic) in varying sizes. Passage of a bougie achieves dilatation with a tangential force.

The main difficulty with bouginage is negotiating the dilator into the stricture and occasionally it may be necessary to do this with a retrograde approach via a gastrostomy.

Bouginage is an effective, quick, cheap and, in experienced hands, safe method of managing esophageal strictures.

Bouginage in children requires general anesthesia. The esophagus is intubated with a rigid esophagoscope and the stricture identified. A lubricated bougie is gently passed through the stricture under direct vision. The diameter of the stricture can be calibrated using the largest bougie which will pass through the stricture without resistance. This should be recorded each time dilatation is performed. It is wise to limit the dilatation in one sitting to a dilator a maximum of three sizes larger than the dilator used to calibrate the stricture. This minimizes the risk of perforation.
Minimal pressure should be applied to the dilator. Excellent results can be obtained.

**Balloon dilation**

Balloon dilation may be safer than bouginage [14]. Balloon dilation has now replaced bouginage as the method of choice for esophageal dilatation in many centers.

Balloon dilation has several merits. A flexible guide wire is negotiated through the stricture using fluoroscopic control. The guide wire remains in place throughout the dilatation which avoids the risk of creating a false passage with the dilator. A balloon dilator applies a radial force to the stricture which, theoretically, should be more efficient.

Progress of the dilatation can be monitored fluoroscopically. The principle disadvantage of balloon dilatation is the absence of tactile feedback during dilatation. Large series now attest to the safety of balloon dilatation in children.

The esophagus is inspected with a flexible endoscope. Once the stricture is encountered an appropriate sized balloon catheter is selected and passed through the working channel of the endoscope and across the stricture.

The position of the guide wire and balloon is confirmed radiologically and visually with the endoscope. The balloon is inflated with a 50:50 mixture of water soluble contrast and saline using a Levene inflating syringe with a threaded plunger and a pressure gauge fitted (Image 2.3). Balloon inflation is observed both by direct vision and radiologically. A waist appears in the balloon as it is inflated which confirms the stricture is engaged. As the balloon is inflated to its rated pressure the stricture is dilated. Successful dilatation is achieved when the waist on the balloon is abolished.

After dilatation the stricture is inspected with the endoscope to exclude perforation. The instrument channel in small pediatric endoscopes is too small to accommodate a balloon catheter. An alternative approach is to cannulate the stricture with a guide wire passed through the endoscope. The endoscope is then withdrawn over the guide wire, which remains in place. This necessitates using a guide wire which is 260 cm long. The balloon dilator is then threaded over the guide wire. The endoscope can be inserted alongside the guide wire and balloon to provide visual confirmation but it is quite acceptable to rely on fluoroscopic guidance.

Although the esophagus is examined with the endoscope after dilation it is wise to perform a chest X-ray and water-soluble contrast study post operatively if there is any suspicion of perforation.

As with bouginage, esophageal balloon dilatation should take place in a progressive fashion. Dilatation should advance in increments of no more than 2 mm. The diameter of the normal esophagus is equal to the diameter of the owner's thumb.

An adequate lumen is usually attained within 6 months to 1 year, with progressively longer intervals between dilations.

Repeated dilatation rarely is successful for the most severe corrosive strictures; early surgical resection is associated with a better outcome.

Esophageal replacement should be considered if dilatation is ineffective beyond 1 year.

**Elective Surgery**

Children failing esophageal dilatation therapy are candidates for surgical reconstruction such as colonic or jejunal substitution, reversed gastric tubes, or gastric pull-up procedures [3, 8].

Primary resection with anastomosis or strictureplasty may be successful for shorter strictures of the esophagus.

Image 2.4 Figures show the stricture before (A) and during (B) balloon dilatation.