Abstract

Objectives: To determine the incidence, management and outcome of esophageal strictures caused by caustic ingestion in children.

Methods: A retrospective review of 83 patients treated for caustic esophageal ingestion during 1985-2004 at King Hussein Medical Center (KHMC). Data were collected: age at presentation, sex, causative agent, incidence of esophageal strictures, complication and outcome were analyzed.

Results: The accidental ingestion of corrosive agents is a major cause of esophageal strictures in children. 83 patients sustained caustic substance ingestion (53 boys and 30 girls). Age at presentation varied from 3 months to 13.5 years, with a male: female ratio of 1.7:1. The majority of caustic substances ingestion was household cleaners (detergents & bleaches) in 41, drain openers (cleaners) in 19. All patients were examined via esophagoscopy in the first 24-72 hours, then by another endoscopy 4 weeks later for follow-up, but in 5 delay patients. The incidence of esophageal stricture was observed in 22.8% (19/83) of the children. The treatment was based on periodic dilatations anterograde (rod) or thread-guided bougies with gastrostomy. Pneumatic balloons have also been employed. Dilations therapy alone was successful in 73.6% (14/19) of patients for 7-36 months duration. There was no mortality and only three esophageal perforations, which did not require surgical treatment.

Conclusion: Caustic esophageal strictures in children can be treated by conservative measure if sufficiently long-term dilatation performed. Esophageal replacement should be considered only in complicated cases or in the rare patients who do not respond to repeated esophageal dilatations.

Keywords
Caustic ingestion, Esophageal strictures, Endoscopic dilatation.

Introduction
Esophageal burn following accidental ingestion of caustic material is seen frequently in children and may lead to life-threatening complications. 1

The potential threat to the children is greater nowadays because of constantly expanding cleaning material market. 2 The ingestion of a caustic agent can initiate a progressive injury to the esophagus, whose extension depends on the type of agent, its concentration, quantity, physical state and the duration of exposure. 3

Most caustic ingestions involve alkalies, esophageal stricture is the most frequent complication and tracheal necrosis is the most frequent cause of death. 4

Esophageal stricture develops within weeks, months or years after alkali ingestion and pyloric stenosis is the most common complication of acid ingestion. 4 Dysphagia is the most common symptom, consequent to esophageal stricture and possible esophageal motility impairment. The severity of dysphagia appears to be more closely related to the degree of alteration of esophageal motility than to the degree of luminal narrowing. 5 The available diagnostic techniques are fibreoptic endoscopy and contrast studies which probably cannot define the depth of the injury. The major determinant in healing or stricture formation is the cicatrix. 6

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In spite of the different treatment methods described for prevention of stricture formation, a considerable percentage of patients develops esophageal and/or gastric stricture, and significant difference of opinion exists in the management of established strictures. However, although surgery represents the only option in cases of acute gastroesophageal necrosis, tracheoesophageal fistula or intractable strictures, the treatment of the vast majority of pediatric accidental caustic ingestion cases, rarely producing these life-threatening complications. A successful resolution can be achieved with periodic dilatations of benign esophageal strictures, and the native esophagus should be preserved if at all possible. The purpose of the present study is to determine the incidence, management and outcome of esophageal stricture caused by caustic ingestion in children.

Materials and Methods

The study was conducted on 83 patients who had ingested caustic material and who were admitted at KHMC between 1985 and 2004. The medical records of the patients were surveyed, included age, sex, type of ingestion, diagnostic studies, treatment methods, complications and outcome.

The ages varied from 3 months to 13 and half years (53 boys and 30 girls). Alkaline substances and particularly drain cleaners (opener) in either liquid or solid form was the offending agent in 9 out 19 cases caused esophageal strictures. Acids also were responsible for 5 esophageal strictures and 1 pyloric stenosis.

Endoscopy is performed with a rigid or a flexible endoscope, within 24 to 72 hours. Five patients were referred to us by other centers for the treatment of esophageal stricture. In acutely burned patients, treatment consisted of starvation, antibiotic coverage, steroids and intravenous fluid during the first 24-48 h.

When the patients were observed to be swallowing saliva, oral fluids followed by milk and milk enriched to provide caloric and protein requirements.

The treatment program for caustic esophageal strictures consisted of 10 to 14 day intervals anterograde dilatations under general anesthesia, starting at 21 post burn day and modifying this protocol as necessary.

In cases of severe caustic burn, as in cases in which the stricture is narrow and tortuous enough to require the use of a guide wire, a gastrostomy was performed with a trans-stricture string (or NGT), followed by anterograde string-guided dilatation of the stricture. Desired final diameter of the esophageal lumen was estimated by the patient's thumb size (5 to 14mm).

Successful periodic dilatation was confirmed by radiological contrast study, allowed for steady lengthening of the interval between dilatation and progress in the maximum size of dilatation.

Results

Among patients who sustained caustic material ingestion and treated at our hospital over 18 years period, 83 enrolled in the study, 53 were males (64%) and 30 were females (36%) with a male: female ratio 1.7:1. The age ranged from 3 months to 13.5 years at presentation. (Fig 1)

Forty-six children (55.4%) ingested alkaline substances, and twenty-four (28.9%) ingested acid agents. The remaining 13, eight cases sustained thermal, neutral and organic material. 5 cases, the nature not completely classified (Table 1).

Accidental ingestion was noted in all cases and no child abuse was established.

Household cleaning products (n=41), drain cleaner (n=19) were found to be the offending ingested materials (Table 1). 57 % (47/83) of the patients endoscopic findings after ingestion appears within normal limits and then were discharged, the remaining 43% (36/83) was found to have aerodigestic tract mucosal injury.

In 33 esophagitis: whole length esophageal burn (n=15), circumferential esophageal ulcer (n=4) and focal or linear erosion (n=14). 57.5 % (19/33) developed esophageal stricture (Table 2).
The offending substances which caused esophageal stricture were drain cleaner in 36.8% (7/19), the remaining 12 cases: acids (n=5), thermal (n=1), organic (n=1), other alkaline (n=2) and in 3 cases the substance was of unknown nature (Table 2).

Regarding the gastric injury, lesions were detected in eighteen cases, varied from hyperemia to prepyloric ulcer. Among these, 16.6% (3/18) stenosis were found in the pylorus. There was no correlation between the intensity of injury of the esophagus and of the stomach.

However, patients who ingested crystalline drain cleaners clinically presented with oral lesions, drooling, vomiting, abdominal tenderness and dysphagia, symptoms almost mimicking those ingested liquid type. Strong acids, in addition, usually presented with upper respiratory tract symptoms such as noisy breathing, choking and cough. So, there is no pathognomonic clinical sign and symptom for the variable caustic materials ingestion (Table 3).

In 5 cases, the diagnosis was missed and the patients were treated in other departments primarily as cases of Mallory Weiss syndrome or angioneuretic edema. Empiric treatment with parenteral nutrition, antibiotics, steroids, histamine-2 (H2) antagonists was immediately applied. Esophageal periodic anterograde dilatation, duration up to 36 months (42 sessions) were needed in some patients with an initial interval of 10 to 14 days starting at 21 day post burn, the frequency then decreased over the first 6-12 months and only 5 patients needed a gastrostomy for thread - guided dilatations. Balloon dilatation was successful in relieving the pyloric stenosis in the three cases of gastric outlet obstruction and helped in other cases in one stage of esophageal dilatation and in cases of severe stenosis, tortuous or multiple strictures.

Among the 19 esophageal strictures, 14(74%) responded successfully to dilatation, the remaining 5 still on ongoing dilatation. When the presentation was delayed for more than one month (n=5), the outcome was significantly less favorable and the patient required prolonged dilatations more than 36 months as in one of our cases. A girl underwent esophageal dilatation up to 3 years then continued once every 3-4 years after recurrent episodes of foreign body impaction for 12 years.

Complications occurred in 35% (29/83) of the patients who ingested caustic agents: esophageal stricture (n=19), pyloric stenosis (n=3), iatrogenic esophageal perforation secondary to blind anterograde dilatation (n=3) with their sequale such as pneumotheorax, effusion and lung abscess. Caustic ingestions were a cause of significant morbidity, but there was no associated mortality. Gastro-Esophageal Reflux (G.E.R) was not severe enough in 4 cases to warrant anti-reflux surgery. But in one case, it might be the cause of recurrent esophageal stricture after a 6- month period of healing and because the medical treatment and follow-up for GER were not taken into consideration.
Fig. 1: Ages of 83 patients with caustic ingestion injuries.

Table (1): Type of ingested material.

<table>
<thead>
<tr>
<th>Substance ingested</th>
<th>No. of patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Household cleaning products</td>
<td>41</td>
<td>49.4</td>
</tr>
<tr>
<td>Drain cleaners</td>
<td>19</td>
<td>23</td>
</tr>
<tr>
<td>Thermal</td>
<td>3</td>
<td>3.6</td>
</tr>
<tr>
<td>Tinner</td>
<td>2</td>
<td>2.4</td>
</tr>
<tr>
<td>N A I S D</td>
<td>2</td>
<td>2.4</td>
</tr>
<tr>
<td>Lye</td>
<td>2</td>
<td>2.4</td>
</tr>
<tr>
<td>H2O2</td>
<td>2</td>
<td>2.4</td>
</tr>
<tr>
<td>HNO3</td>
<td>2</td>
<td>2.4</td>
</tr>
<tr>
<td>HCL</td>
<td>1</td>
<td>1.2</td>
</tr>
<tr>
<td>H2SO4</td>
<td>1</td>
<td>1.2</td>
</tr>
<tr>
<td>Car shampoo</td>
<td>1</td>
<td>1.2</td>
</tr>
<tr>
<td>Alkaline gel</td>
<td>1</td>
<td>1.2</td>
</tr>
<tr>
<td>Kerosine</td>
<td>1</td>
<td>1.2</td>
</tr>
<tr>
<td>Unknown</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>83</td>
<td>100</td>
</tr>
</tbody>
</table>
Table (2): Endoscopic findings of esophageal caustic injury.

<table>
<thead>
<tr>
<th>Findings</th>
<th>No. of cases</th>
<th>Alkaline</th>
<th>Acid</th>
<th>Others</th>
<th>Normal findings 4 weeks later</th>
<th>Persistent esophageal stricture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole length esophageal burn</td>
<td>15</td>
<td>10</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td>Circumferential esophageal ulcer</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>1 (Alkaline)</td>
<td>3</td>
</tr>
<tr>
<td>Focal / linear esophageal erosion</td>
<td>14</td>
<td>7</td>
<td>5</td>
<td>2</td>
<td>10 (5 Alkaline)</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>33</td>
<td>20</td>
<td>10</td>
<td>3</td>
<td>12</td>
<td>19</td>
</tr>
</tbody>
</table>

Table (3A): Clinical presentation of patients with caustic material ingestion.

<table>
<thead>
<tr>
<th>Substance</th>
<th>Main symptoms and signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Car shampoo</td>
<td>Hematemesis. Constipation.</td>
</tr>
</tbody>
</table>

Table (3B): Clinical presentation of patients with caustic material ingestion.

<table>
<thead>
<tr>
<th>Substance</th>
<th>Main symptoms and signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>HNO3</td>
<td>Harsh breathing. Drooling. Dysphagia.</td>
</tr>
<tr>
<td>Hype</td>
<td>Vomiting</td>
</tr>
<tr>
<td>H2O2</td>
<td>Epigastric pain</td>
</tr>
<tr>
<td>HCL</td>
<td>Not significant!</td>
</tr>
<tr>
<td>Flash</td>
<td>Not significant!</td>
</tr>
<tr>
<td>Alkaline gel</td>
<td>Vomiting. Abdominal pain.</td>
</tr>
</tbody>
</table>
Discussion

Accidental ingestion of caustic chemical by children still represents a major problem. Household solutions of detergents or bleach are the most common ingested materials. Drain cleaners containing NaOH and/or KOH are the principal causes of corrosive esophageal injury. It is known that ingestion of alkali results primarily in oropharyngeal and esophageal damage, while acids tend to spare the esophagus and produce coagulation necrosis of the stomach as in a thermal burn.

The degree of mucosal injury depends on the amount and concentration ingested, the duration of contact with mucosa, position and mode of ingestion, the nature of the agent and whether it was taken in fasting or fed state at the time of ingestion. According to shikowitz et al, many variables affect the incidence of stomach injuries, such as tonus of the pylorus and type of food ingested before the caustic agent. Initial management post burn is directed towards establishing the diagnosis, assessing the severity of injury and maintenance of an adequate esophageal lumen. Gastric lavage or emetic are contraindicated because these increase the risk of perforation or aspiration and re-exposure to the caustic agent. Neutralization is contraindicated also, because of its impracticality and potential exothermic reaction.

Endoscopy should be credited as a life saving procedure in the presence of black discoloration indicating mucosal gangrene with full thickness necrosis. Early esophagoscopy, within 24-36 hours after the injury is extremely important for assessing both the degree of esophageal burn and the protocol for subsequent treatment. Delay initial endoscopy for 24 hours and 48 hours allows the full extent of the damage to manifest and within 4 days of the injury before the esophageal wall begins to weaken substantially.

Contrast esophagography is not helpful in the early stages of acute injury unless there is strong suspicion of perforation, or it is anticipated that endoscopy cannot be performed for some reason, but 2 weeks after ingestion is of great value to elucidate the extent of stricturing to allow assessment of dynamic esophageal function and at regular intervals to assess results following a dilatation. If there are no abnormal findings at esophagogastroscopy, the patient is discharged but must be followed up for at least 1 month. Authors recommended follow up regularly at 6- month intervals with both esophagogastroscopy and esophagography for 1 year.

If erythema or spotty ulceration, the patient is admitted and antibiotics, steroids and H2 blocker are administered for 14 days together with general measures. If circumferential ulcerations are recognized, these measures continued for 21 days. Additionally, a stent (NGT/String) must be inserted into the stomach. The stent apparently prevents cross-fusion of the adjacent damage areas as well as obstruction of the esophageal lumen due to oedema and excessive granulation tissue.

Fibroblast proliferation within the wound is followed by collagen deposition over the first week. The burn area is the weakest between 7 and 21 because of sloughing of necrotic tissue and new collagen formation. Various factors are responsible for stricture development, including obliteration of the esophageal lumen by oedema, excessive granulation tissue, adhesions between adjacent ulcerated areas, contraction of the fibrous scar formed in the esophageal wall and destruction of the myenteric plexus. When strictures develop, they reflect the severity of the initial injury rather than measures taken in the emergency period. Fatty et al. reported that scar formation at the site of corrosive esophageal burns occurs 2 or 3 weeks after injury and that esophageal strictures developed in 58% of patients by 1 month, in 80% by 2 months and in 99% by 8 months.
Several studies have indicated that clinical manifestations of caustic ingestion injuries are poor predictors of the extent or depth of esophageal injury. \(^\text{14}\)

Ashcraft and Simon have pointed out that no uniform criteria are available for the evaluation of burn severity by endoscopy. \(^\text{15}\)

The sequelae of caustic ingestion are not limited to esophageal stricture and its direct consequences but also include severe disturbances of esophageal motility which in turn cause dysphagia, choking attacks, GER, impaction of food that were traditionally attributed to stricture. \(^\text{5, 22}\)

After the caustic ingestion, the earliest recognition of Gastric Outlet Obstruction (GOO) varies from 7 days to 3-5 years. Late sequelae of corrosive gastric injury, duodenal atony, mucosal metaplasia and gastric carcinoma. \(^\text{12}\)

The mainstay of treatment in established esophageal stricture has been graded dilatations, whether anterograde or retrograde, commencing 3 weeks after the injury and performed at 10–14 day intervals for 6 to 12 months after the initial insult or until fibrosis is no longer progressive. \(^\text{8}\)

The optimal frequency and time of such procedures is not well established and is largely individualized. The time interval between procedures is based on the effects of the previous dilatations and symptomatology. \(^\text{14}\)

Experimental and clinical surgical evidence shows that scar tissue progressively contracts up to six months after the start of the healing process. Therefore, continuous dilatation has to be performed for a long time with a range from 6 months to twelve years, or even 14 years after their initial injury. \(^\text{16, 17}\)

Balloon dilatation could be used preferentially in very narrow strictures; it may be used in strictures with tortuosity of the esophagus and in cases of G.O.O because of a possibly smaller risk of perforation. \(^\text{16}\) In some cases, severe scar formation might limit the effectiveness of balloon dilatation, since Fibrosis with altered blood supply may reduce elasticity of the tissue. \(^\text{7, 16}\)

The main disadvantage of conservative treatment by periodic dilatations consists in the large number of dilatations often needed to achieve complete healing of the stricture. \(^\text{9}\) But, conservative procedures based on the fact that a burned but conductive esophagus is better than any substitutive plasty, even if it is done well there will still be problems. \(^\text{2, 17}\)

Daly and Cardona described the indication for esophageal bypass to be: complete stenosis with failure to establish lumen, the presence of a fistula, marked irregularity and pocketing of the esophagus. \(^\text{8}\)

In our retrospective study over a period of 18 years, 83 children were admitted to our hospital with a history of caustic ingestion. Ages ranged from 3 months to 13.5 years. Sixty-one (73%) of the patients between the ages of 1-4 years (Fig 1).

Esophageal stricture is the most frequent and significant long-term complication, it was found in 22, 8% (19/83) of cases (Table 2). The offending ingested substance was an alkali in 70% (56/83) of children, the most common agent was drain cleaner in 22, 8% (19 patients).

None of our patients carried severe gastric or esophageal damage or delayed gastric syndrome. Crystalline drain cleaners in the form of concentrated sodium hydroxide tend to the oropharynx or become lodge in the upper esophagus where injury is most severe while those in a liquid form produce a more diffuse injury pattern. \(^\text{18}\) However, 2 out of 4 who ingested crystalline drain cleaner endoscopically were found to have whole length esophageal mucosa injury. Thus, the fact that ingestion caustic fragments does not eliminate the possibility of stenosis, even of a severe grade. \(^\text{12, 19}\)
The majority of strictures treated effectively in 14 out of 19 cases by repeated dilatation, during 7 and 36 months. The remaining 5 cases are on going dilatations.

Time delay was more than 3 months for a girl who had GER, after 24 months duration of dilatation, she was free for six months then she came with esophageal stenosis again.

However, a strong predictor of poor outcome, time delays from ingestion to commencement of dilatations, gastroesophageal reflux which has adverse effects in already severely burned contracted esophagus. Recurrent symptoms observed long after the termination of follow-up in one of our patients was attributed to the recurrent stricture in presence of gastroesophageal reflux disease. So, every caustic esophageal burn patient should be screened for GER at least once every 3 months with distal esophageal 24-h pH-metry. H2 antagonist and antacids should be considered in the management of these injuries, some patients may require anti-reflux procedures, for achieving faster resolution of strictures and may also contribute to the reduction of the well-known risk of squamous cell carcinoma development in the burned esophagus. The incidence is 0.8-4% and age for the diagnosis ranged from 13-71 years. Since the evaluation of clinical symptoms may be subjective in children, loss of symptoms during follow-up never played a determining role in the termination of follow-up without radiographic documentation of esophageal healing. Endoscopic dilatation of the stricture leads to complications in up to (3-6 perforation per 1000 dilatations) and should not exceed (0% to 1, 4%) in expert hands. In our study, this complication reached up to 15, 7%. The most obvious risk factors are anatomic abnormalities that preclude safe unguided dilatation. So, bougies for antegrade dilatation should never be passed blindly, retrograde dilatations are the safest and most successful method of therapy for severe stricture. However, with the appropriate management, a good outcome can be expected for patients with iatrogenic esophageal perforation. Mortality is 0%. Surgical management is not required in 3 of our patients because of this condition.

As noted by key, authors believe that superficial injuries heal without any consequence. If the damage is not circumferential, then the likelihood of stricture formation is remote, it is not the case in our study (Table 2). Even though gastric outlet obstruction is a well recognized sequale of corrosive ingestion, it is uncommon and has been associated with esophageal stricture in about 20% of cases. Among our patients, 3 (15.7%) cases of GOO was identified, the causes were acid ingestion, hot water and unknown chemical ingestion, respectively; all were treated successfully by balloon dilatation.

In our experience, conservative treatment of the burned esophagus is almost always feasible. Comparable with those reported in the literature. Conclusion we believe that most caustic esophageal strictures in children can be treated by a conservative measure if a sufficiently long-term dilatation performed, esophageal replacement should be considered only in complicated cases or in the rare patients who do not respond to repeated esophageal dilatations. Special care should be given to protect children from accidental caustic ingestion.
References


دراسة حول اصابة المريء بواسطة تناول المواد الكيميائية

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ملخص:

أجريت هذه الدراسة على 83 طفلاً حيث ادخلوا إلى مدينة الحسين الطبية في الفترة ما بين 1985 إلى 2004 بعد تناولهم بشكل غير مقصود للمواد الكيميائية ذات الاستخدام المنزل في حيث كانت أعمارهم تتراوح بين 3 أشهر إلى 13.5 سنة.

اغلبية تلك المواد كانت مواد تطبيب أو مواد لفتح المصارف المنزلية مما أدى إلى حدوث مضاعفات أغلبها كانت تضيق المريء أو انغلاقه عند ما يقارب 23% من الأطفال. مضاعفات وأثر تناول هذه المواد قد تظهر مباشرةً أو بعد مضي أسابيع أو أشهر أو حتى سنوات. إن اجراء تطبيب للمريء والمعالجة في الفترة ما بين 24 إلى 72 ساعة بعد اساسياً بالإضافة إلى الصور الشعاعية الملونة.

عند كل من المهم جداً وضع بروتوكول للعلاج من ذك، التشخيص الاصابة والبدء في حال مستعد إصابته النازح بعد إشارة إلى فترة قد تصل 36 شهراً وفلا فائت النتيج المرضى تكون ضئيلة.

المفتاح الكلمات:

تناول مواد كاوية، تضيق المريء، الموسعت.