The Study of Effects Regarding Ingestion of Corrosive Substances in Children

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Corrosive substances, acids or alkaline, cause tissue lesions, by means of chemical reactions. The most frequent complication of corrosive esophageal lesions is esophageal stricture. We conducted a retrospective study on a batch of 115 children, admitted in the V-th Clinic of Pediatrics, St. Mary Children Emergency Hospital, for ingestion of corrosive substances. High frequency was noticed in the small ages (1-4 years old) 94 cases (81.74%). Out of the 115 cases, 46 complicated with esophageal stricture, which were either dilated with Savary devices (42 cases), or underwent surgery (4 cases).

Keywords: corrosive substances, acid, base and oxidant solutions, esophagitis, child

Corrosive esophagitis is one of the most frequent forms of esophagitis in children and is found to a larger extent in the rural environment, where the supervision of small children is faulty. The ingestion of corrosive substances in children is usually accidental [1]. Voluntary ingestion is found mostly in suicidal adolescents. In the United States, studies reported approximately 5,000 – 15,000 new cases a year [2, 3].

The toxic substances cause destructive lesions of various depths in the upper digestive tract walls, up to their perforation. Corrosive agents with a *p*H < 2 or > 12 quickly penetrate the esophagus layers and lead to scar tissue due to necrosis, which limits the deep penetration of tissues [4].

The ingestion of corrosive substances can cause lesions in the lips, oral cavity, pharynx and upper airways. The respiratory system is mostly affected especially due to its exposure to corrosive vapors. In children, 18 to 46% of all ingestions of corrosive substances are associated to esophageal lesions [5].

Sometimes, in children, there are no signs and symptoms suggesting corrosive esophageal lesions, therefore some studies propose the analysis of predictive biochemical markers for the ingestion of corrosive substances and for the complication of preexisting lesions.

The gravity of lesions is determined by certain factors: the nature of the substance (acid, base or oxidant), pH, ingested amount, form of presentation (liquid corrosives cause more extended lesions than solid ones), duration of the contact with the corrosive substance, stomach repletion or depletion, the time of reaching the hospital, the time of the original endoscopic examination, the time of the decision on surgical or instrumental correction, and the time of the initiation of pharmaceutical treatment [6, 7].

Corrosive substances cause tissue lesions by means of chemical reactions. These substances can be acid or alkali. The most important lesions are produced by acids with a *p*H less than 3 or by bases with a *p*H greater than 11 [8].

Acids, by denaturing superficial tissue proteins, cause coagulation necrosis generating scar tissue. Strong acids have immediate, almost instantaneous effect. Scar tissues have a protective role, which limits the penetration of deep layers. The stomach and lower esophagus are affected most frequently. The denudation of the scar tissue exposes to a major risk of perforation. Pyloric stenosis often occurs. The acids in commercial products are represented by the hydrochloric acid, sulfuric acid and silver nitrate [9]. They are found in the bathroom, pool and rust cleaning products [10].

Base ingestion causes liquefactive necrosis with quick and severe tissue penetration. Lesions are generated by fat saponification, denaturation of proteins and vascular thrombosis [9, 10]. Even low concentrations can produce extended prejudice. A study conducted in Brazil reports the fact that a solution with 1.83% concentration can cause esophageal necrosis within an hour [11]. The oropharynx and the esophagus are affected most often. Severe gastric damage is not rare. The complications of base ingestion are represented by esophageal strictures and fistulas, and posterior cricoarytenoid muscle fibrosis. There is a risk of malignant transformation of fibrous lesion. Basic substances are represented by sodium hydroxide (caustic soda), potassium hydroxide, sodium carbonate (washing soda) and potassium carbonate.

Other corrosive substances are represented by some oxidants used in households, like: sodium hypochlorite and potassium hypochlorite, hydrogen peroxide, iodine and its compounds (organic and inorganic), formalin, phenol, ammonia, zinc and mercury chloride.

From the anatomo-pathological point of view, the lesions caused by corrosive substances in the digestive tract evolve to 3 stages: acute – immediately after the ingestion (day 1 – 4) generating erythema, edema and intense cellular necrosis; latent (day 5 – 15) generating cellular edema, ulcerations and granulation tissue; chronic (after day 15) generating fibrosis and scars with stenosis. The maximum

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Degree 0	Normal endoscopic image
Degree I	Erythema and edema limited to the mucous membrane [± small non-bleeding erosions]
Degree II a	Friability, erosions, superficial ulcerations, whitish membranous exudate
Degree II b	Deep or circumferential ulcerating lesions
Degree III a	Deep hemorrhagic ulcerations, small necrosis areas
Degree III b	Extensive necrosis
Degree IV	Perforation

Modified according to Zargar et al. [12]

Table 1
ENDOSCOPIC CLASSIFICATION OF CORROSIVE ESOPHAGEAL LESIONS

risk of esophageal perforation is present during the first 3 days from ingestion and between days 6 to 10.

Experimental part

Material and method

We conducted a retrospective study on a batch of 115 children of both genders, coming both from the rural and from the urban environment, aged 1 to 18 years, admitted in the Vth Pediatrics – Gastroenterology Clinic of *St. Mary* Children Emergency Hospital, Iasi, Romania, for ingestion of corrosive substances.

In case of ingestion of corrosive substances, the upper gastrointestinal endoscopy (UGIE) is performed as early as possible (the first 48 h, preferably during the first 12 h), immediately after the patient is stabilized. This allows the exact mapping of the lesions and the exact determination of the esophagitis degree (table 1). UGIE determined the reduction of mortality by approximately 50% during the past 25 years. It is the most efficient method of monitoring the evolution. It plays an important role in prognostic orientation and in the dilative or protein therapy of stenosis. It plays an essential role in establishing the type of medical treatment and in the indication of surgical treatment.

The treatment of esophageal stenosis – complication of corrosive esophageal lesions – consists in successive esophageal dilations using Savary bougies (fig. 1) or balloons [13].

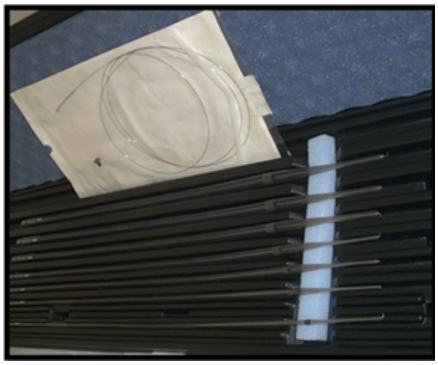

Fig. 1. Savary bougies

The equipment necessary for esophageal dilations is made of dilators, guiding wire and an endoscope. The ideal dilator must easily pass through the oropharynx and the stenotic area, allow the physician to feel the stenosis, require minimum passages through the pharynx, have a radio-opaque marker, be usable in case of hiatal hernia or an operated stomach, pass on a guiding wire with imperfections, and be cost-efficient. The dilation technique is similar for all types of dilators. The basic principle consists in introducing through the stenosis dilators of increasing sizes. The dilation with Savary bougies is performed in left lateral decubitus. The guiding wire is passed through the biopsy channel under direct vision into the gastric antrum. If the stricture area cannot be surpassed with the endoscope above it, the guiding wire is introduced through the biopsy channel and through the stenosis and is placed under radiologic control into the stomach (the diaphragm is used as a marker). After placing the guiding wire, the endoscope is gradually retracted and simultaneously the guiding wire is introduced a few centimeters further through the endoscope. The proper dilation procedure begins by introducing the smallest dilator and continuing until resistance is felt. The Savary probes have a radio-opaque marker and ease radiologic monitoring of the correct introduction in special cases.

In moderate stenosis, progressive endoscopic dilations were applied by means of Savary bougies (fig. 2).

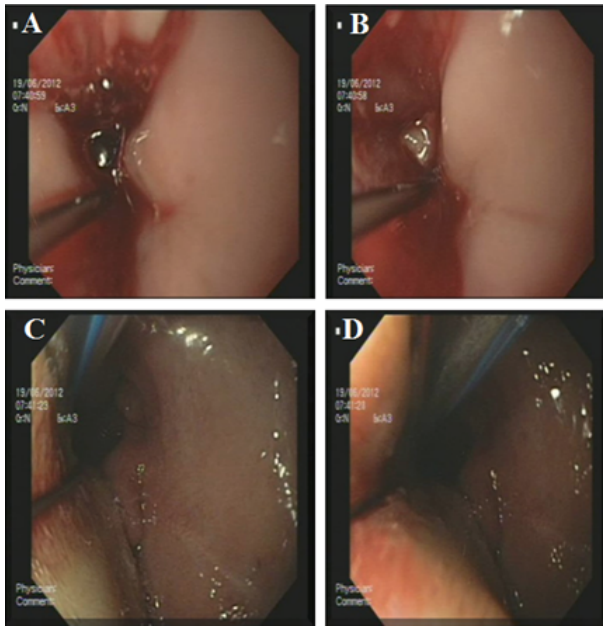

Fig. 2. Dilations with Savary bougies

Results and discussions

All children with post ingestion esophagitis followed corticoid therapy for one month and antibiotherapy. They were evaluated by UGIE at 1 month and at 6 months to notice the esophagitis' evolution and eventually the esophageal stenosis.

The ingestion of corrosive substances was more frequent in small ages (1-4 years old) 94 cases (81.74%).

Regarding the esophagitis degree, according to Zargar classification, most frequent was: type I esophagitis 57 children (49.56%), type II esophagitis – 33 children (28.69%), type III esophagitis – 25 children (21.73%).

Out of the 115 cases of corrosive esophagitis, 46 cases complicated with esophageal stricture.

Out of the 46 cases of esophageal stenosis, 4 cases underwent surgery (in 3 cases a digestive tract graft was performed, and in 1 other case a left colon graft was performed).

The distribution of children with esophageal strictures was: small children (1 – 3 years) – 33 children (71.74%), in big children (4 – 13 years) – 9 children (19.57%), and in adolescents (14 – 18 years) – 4 children (8.69%). Their distribution according to gender showed a slight

preponderance of females: 25 (54.35%) girls and 21 (45.65%) boys. As regards their environment of origin, the rural environment prevailed: 71.74% cases and 28.26% cases from the urban environment.

The polymorphic clinical picture depends on the lesions caused by the ingestion of corrosive substances, as well as on the individual intrinsic activity and is represented by pain in the digestive segment in question (oropharyngeal, retrosternal, epigastric), of variable intensity, depending on the depth of the lesions, heartburn, dysphagia, sialorrhea, dysphonia, nausea, vomiting, hematemesis and melena. Dyspnea appears due to laryngeal edema or pulmonary aspiration, and stridor appears due to glottal edema. Sometimes, these require tracheotomy and are usually associated to extended esophageal lesions [14]. Anxiety or psychomotor agitation can also appear.

General status varies depending on the gravity of lesions. An examination of the oral cavity can reveal hyperemia, edema and ulcerations. Fever can appear in medium and severe forms and is a gravity indicator. Abdominal muscle contraction can be present in gastric perforations [9].

The relation between symptoms and the gravity of lesions is uncertain [15]. In specialized literature, the incidence of coexisting gastric lesions is reported as being between 20% and 62% and varies from simple hyperemia to diffuse transmural necrosis erosions [16, 17]. Immediate or delayed gastric perforation is associated to high mortality and is much more rarely reported in children than in adults [18].

Biochemical changes following the ingestion of corrosive substances have not been studied in detail in the specialized literature. Leukocytosis (>20,000/ mm^3), the presence of the inflammatory syndrome and esophageal ulcer have been associated to a high mortality rate in adults [19]. Arterial *p*H less than 7.22 or an excess of bases lower than -12 were considered an indicator for severe esophageal lesions associated to emergency surgery [20].

Simple abdominal X-ray can show the presence of radiopaque pills, gastric dilation, ileus or pneumoperitonemum.

A computed tomography scan is the most sensitive method for the early detection of perforation.

The treatment immediately following the ingestion of corrosive substances implies respiratory, cardiovascular, renal, electrolyte, and acid-basic monitoring. No food should be administrated during 12 hours. Painkillers and tranquilizers can be used. There are contradictory studies regarding the administration of corticoid therapy. Antibiotherapy is used as prophylaxis. Anti-secretory drugs can also be administrated: proton-pump inhibitors.

Progressive endoscopic dilations by means of Savary bougies were performed in 42 cases of moderate esophageal strictures and 4 cases underwent surgery.

Complications of esophageal stenosis dilations are rare. No post-dilation discomfort should be neglected. Perforation is the most severe complication. Generally, it occurs at a level close to the stenosis, in the case of the balloon method. Post-dilation hemorrhage, aspiration pneumonia or bacteremia can also emerge. The rate of perforations after the dilation of corrosive stenosis varies between 0.4% and 32%., decreasing to 17.6% - 4.5% as the experience of the personnel is higher [21]. We had no case with perforation.

When esophageal dilations are not possible or an appropriate long-term esophageal caliber cannot be ensured, surgical treatment is recommended. It is also recommended in digestive tract perforations. Mortality and morbidity are low when the surgeon is experienced [22].

Conclusions

Esophageal strictures occupy an important place in pediatric pathology, with multiple implications. Caustic soda ingestion is one of the most invalidating intoxications, as in over 90% of the cases esophageal stricture sets in and seriously affects life quality. Dilations with adjustable Savary bougies with metallic wire endoscopically guided and calibrated – a modern method of solving esophageal stenosis, especially corrosive ones – diminish as much as possible the perforation risk and allow the avoidance of surgery in many cases. Using this kind of treatment, the evolution towards healing of corrosive esophageal stricture is possible.

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