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Corrosive Poisonings in Adults

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REVIEW

SUMMARY

Ingestion of corrosive substances may cause severe to serious injuries of the upper gastrointestinal tract and the poisoning can even result in death. Acute corrosive intoxications pose a major problem in clinical toxicology since the most commonly affected population are the young with psychic disorders, suicidal intent and alcohol addiction. The golden standard for determination of the grade and extent of the lesion is esophagogastroduodenoscopy performed in the first 12-24 hours following corrosive ingestion. The most common late complications are esophageal stenosis, gastric stenosis of the antrum and pyloris, and rarely carcinoma of the upper gastrointestinal tract. Treatment of the acute corrosive intoxications include: neutralization of corrosive agents, antibiotics, anti-secretory therapy, nutritional support, collagen synthesis inhibitors, esophageal dilation and stent placement, and surgery.

Key words: corrosive substances, esophageal injuries, sodium hydroxid, postcorrosive stenosis , esophagogastroduodenoscopy.

1. INTRODUCTION

According to the report of the American Association of Poison Control there are about 200,000 caustic poisonings annually, most frequently with acid and alkaline agents that are used as cleansing substances in the households (1, 2). Data about the Republic of Macedonia give evidence of about 75-80 corrosive poisonings per year, more frequently found in women and in 95% of the cases they are with suicidal intent (3). Depending on the degree of post-corrosive injuries, stenosis along the entire esophagus appears, especially in distal and middle third of the esophagus, at the level of physiological narrowing and in the gastric antrum and pylorus (4, 5). The intoxication may lead to death, more often in the acute phase although unfavorable outcomes have been described in all phases of the poisoning. Esophageal carcinoma may develop as a late complication, which onset has been observed 40-60 years after caustic ingestion (6, 7, 8).

2. CORROSIVE SUBSTANCES

Oral intoxication with corrosive agents occurs by ingestion of: acids (hydrochloric, acetic, sulfuric, lactic, oxalic, carbolic), alkalis (sodium and potassium, soaps, detergents), heavy metal salts (sublimate), formalin, iodine tincture and many other chemical substances. Lye is a general term in the American literature, denoting strong alkali found in cleansing agents (9, 10).

2.1. Acids

In our environment the most common abused acid is hydrochloric acid (more than 50%), which is easily accessible as a sanitary cleansing agent. It is often used in countries like India and Taiwan as opposed to the USA where its abuse is less than 5%. It usually causes gastric stenosis although cases of esophageal stenosis have also been described (11). Beside intoxication with hydrochloric acid, intoxication with sodium hypochlorite has been observed ($NaClO_4$, about 20% of the total number), which as a solution or in combination with hydrochloric acid is used for cleaning sanitary facilities and swimming pools. It rarely causes severe injuries of the upper gastrointestinal tract. Rarely observed are also severe injuries with acidic acid (acidum aceticum glaciale–CH_3COOH, about 11% of the total number), which is used in the food industry for vegetables conservation and is often abused during the season of preparing food for winter. There are also rare poisonings by ingestion of chemical agent in battery fluids (sulfuric acid – H_2SO_4), household bleaches (5% of Na hydrochloride), antirust compounds (oxalic acid), hydrogen peroxide that is used as a cosmetic agent in a concentrated form (12).

2.2. Alkalis

Beside acids, corrosive alkalis are also being abused, such as sodium hydroxide (NaOH) and potassium hy-

droxide (KOH). Their abuse is more frequent in the USA and member states of the European Union and they are found on the trade market as fluids and paste or granular forms. They have a high Ph value, ranging from 4% to 54%, and are found as components in the detergents, soaps, cleansing tablets and cosmetics. They are used in everyday life for cleaning sanitary surfaces and as drain openers. These substances may cause severe post-corrosive injuries of the upper gastrointestinal tract, including perforation that often results in death. The most common complications are esophageal and gastric stenosis, which are found in greater percentage than in poisonings with acid substances (13, 14).

2.3. Pathophysiology and pathology

In contact with acids, tissue proteins are transformed into acid proteins and hemoglobin is transformed into hematine. The final outcome is the so-called coagulation necrosis.

In contact with alkalis, tissue proteins are transformed into proteinates and fats into soaps, resulting in penetrating, that is liquefaction necrosis (15).

Corrosive substances with a Ph of less than 2 or greater than 12 are highly corrosive and can cause tissue necrosis. A concentrated solution of sodium hydroxide (22.5% and 30%) in contact with the esophagus can produce perforation of the esophageal wall, mediastinitis and fatal outcome for 10 seconds or for 1 second (16). The severity of the chemical burns that affect the entire gastrointestinal tract depends on several factors: nature of the corrosive substance, Ph value, the quantity and concentration ingested, duration of exposure and the act of swallowing. The physical characteristics of the corrosive substances (fluid or solid form, gel or granules) might influence on the localization of the post-corrosive injury. Ingestion of corrosive substance in a solid or gel form causes injuries at the level of the oropharynx and proximal segment of the esophagus, while corrosive liquid substances cause injuries on the middle and distal segments of the esophagus and stomach (17).

Few hours after corrosive ingestion, thrombosis of small vessels appears, producing heat that exacerbates the injury. These processes in the esophageal wall and stomach continue in the next several days when bacterial invasion occurs as well as the so-called inflammatory response and development of granulation tissue (18). Consequently, collagen deposition is minor until the second week after ingestion, and the healing process begins three weeks after ingestion. Three weeks after ingestion, tissue fibrosis occurs, resulting in narrowing of esophageal and/or stomach lumen and stricture formation (19).

The pathologic classification of corrosive injuries of the upper gastrointestinal tract is similar to the classification of thermal skin burns. First degree: is characterized by superficial damage followed by onset of mucous edema and erythema. The affected mucous layer regenerates in a few days and usually does not manifest complications such as scars or stricture formation.

Second degree: is characterized by caustic penetration through the submucosa into the muscular layer of the organ. After one to two weeks, deep ulcerations and granulation tissue develop on the esophageal or gastric wall. Additionally fibroblast reaction ensues, production of collagen tissue that loses its humidity and is subjected to contraction over a period of several weeks or months. These processes along with the neighboring injuries may cause narrowing of the esophageal or stomach lumen within the next 8 weeks to 8 months and stricture or stenosis may appear. Esophageal stenosis most frequently develop at the cricopharyngeal area, at the level of the aortic arch and tracheal bifurcation and the lower esophageal sphincter. Most gastric stenosis occur in the atrium and pylorus.

The third degree is characterized with perforation of the wall of the esophagus or stomach (20).

3. CLINICAL CHARACTERISTICS

Clinical presentation of corrosive injuries in the upper gastrointestinal tract depends on the physical state, type and quantity of the corrosive substance. After caustic ingestion patients complain on painful and burning mouth and throat, retrosternal chest and stomach pains, nausea, vomiting, often with bloody content. These symptoms may develop immediately after caustic ingestion, or be delayed for few hours after ingestion and they may last days and weeks. Hypersalivation, difficulty in swallowing with edema, ulceration or whitish plaques in the oral cavity, palatal mucosa and pharynx are common phenomena (21).

Corrosive substance ingestion in the acute phase may result in injuries of the larynx and may cause laryngospasm associated with dyspnea, tachypnea, dysphonia and aphonia. Aspiration of the corrosive substance may cause endotracheal or bronchial necrosis with mediastinitis, often leading to fatal outcome (22).

Local obvious lesions are painful and contact bleeding. Presence of hoarseness induces laryngeal, epiglottal or hypopharyngeal complications. High temperature accompanied with fever induces perforation and suggests consultation with a surgeon. The absence of oropharyngeal changes does not preclude severe injuries of other segments of the gastrointestinal tract. In 10% to 30% of patients with severe esophageal post-corrosive burns there are no local changes in the oropharynx. One extensive study reported on 37% of esophageal injuries of second and third degree in patients who had no apparent oropharyngeal injuries (33, 34). On the other hand, other studies showed that 70% of patients with severe oropharyngeal injuries did not have significant esophageal post-corrosive burns. Therefore, injuries of the orpharynx are not a reliable indicator for the eventual damage to the esophagus (24).

Loss of large quantity of liquids and metabolic complications (acidosis) along with renal failure even more complicate the general condition of the patient. Severe caustic injuries of the stomach may result in perforation of its wall and development of acute abdomen, which requires emergency surgery. These injuries may appear in the first 48 hours or they may be delayed until the 14th day after corrosive ingestion (25). (Figure 1).

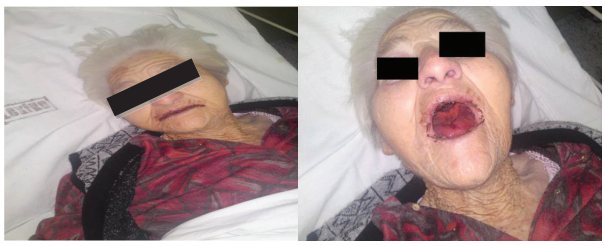

Figure 1. Patient with severe local post-corrosive injuries

4. POST-CORROSIVE LATE COMPLICATIONS

Post-corrosive late complications are a major problem in acute corrosive poisoning and often cause difficulties in everyday activities of the patients. Sophisticated diagnostics and contemporary protocols for treatment have reduced the percentage of post-corrosive late complications related to acute corrosive intoxications of the upper gastrointestinal tract. However, they still pose a serious medical and social problem, both by their clinical presentation and therapeutic approach.

The most common late complications are esophageal strictures and stenosis, gastric stenosis of the antrum and pyloris, esophageal and stomach cancer (26, 27).

Strictures and stenosis of the esophagus – difficulty in swallowing and a feeling of pressure behind the sternal bone are the most common symptoms indicating narrowing of the esophagus. They may appear three weeks after ingestion of the corrosive substance, in the first three months or, according to some authors, even after one year following caustic ingestion. Liquid corrosive substance ingestion more often initiates stenosis than corrosive substances in crystal form. (Figure 2).

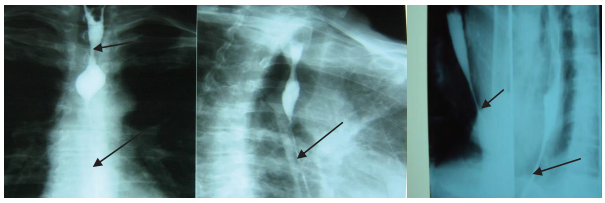

Figure 2. Post-corrosive narrowing of the mid and low part of the esophagus

Stenosis of antrum and pyloris – onset of symptoms of full stomach, nausea, vomiting, and weight loss suggest gastric obstruction. It occurs rarely than esophageal stenosis, often 5 to 6 weeks after the ingestion and, according to some authors, it may appear even after several years. It is most frequently found after acid ingestion although many cases of gastric stenosis have been reported after alkaline ingestion (Figure 3).

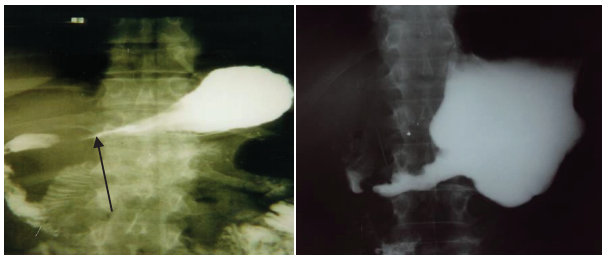

Figure 3. Antropyloric and pyloric post-corrosive gastric narrowing

Esophageal and stomach carcinoma – development of carcinoma might happen 40 to 50 years after ingestion of the corrosive substance. According to some studies, 3% of patients with esophageal carcinoma presented with a history of previous caustic substance ingestion. Most lesions occur at the level of the carina. Gastric carcinoma is a very rare complication after corrosive ingestion (28, 29).

5. MANAGEMENT

Prehospital procedures – gastric lavage, induced vomiting and activated charcoal are contraindicated because re-exposure of the esophagus to the corrosive agent might happen and produce additional injuries. Milk and water are suggested to be useful in the acute phase (the first 1-3 hours) but their effectiveness has not been proven in controlled studies. Milk may compromise urgent esophagogastroduodenoscopy and the heat produced during the chemical reaction might cause additional post-corrosive injuries (30).

Radiologic studies – in the acute phase, a plain radiography of the chest and abdomen may give useful data about the dimensions of the mediastinum and may reveal air in the mediastinum or under the diaphragm suggesting esophageal or gastric perforation. Esophagogastroduodenography with gastrographin 25 – 30 days after corrosive ingestion may give us useful information about the changes in the dimensions of esophageal and gastric lumen. Some authors prefer barium sulfate as less irritant, especially if it is aspirated and it can also be immediately used after ingestion for monitoring the development of complications. This procedure is not suggested in the acute phase except if there is an absolute contraindication for esophagogastroduodenoscopy or suspicion of eventual perforation (31, 32).

Esophagogastroduodenoscopy – is a sophisticated and sovereign method for diagnostic evaluation of acute corrosive intoxications and lesions of the upper gastrointestinal tract. There is a unanimous agreement that the most optimal timing for esophagogastroduodenoscopy is the first 12 – 24 hours post-ingestion. Since inflammatory changes, vascular thrombosis and the healing process of the post-corrosive injuries begin the 4th and are most intensive until the 14th day, it is suggested to avoid this diagnostic procedure during this period (33).

Unfortunately, there are still controversial opinions about the timing, and method of performing this procedure. Some authors suggest that all patients with distinct symptoms of caustic ingestion have to be subjected to endoscopy. On the other hand, endoscopy should not be performed in asymptomatic patients with absence of oropharyngeal injury, but they should be rather observed for 48 hours in hospital settings. However, the fact that even 20% of post-corrosive poisonings pose no signs of oropharyngeal injury as well as the unproved correlation between the symptomatology and the severity of post-corrosive injuries advocate a legitimate need, with no exception, of upper endoscopy in all poisonings with caustic substances (34). The latest trials suggest an

eventual urgent upper endoscopy to be carried out after previous sedation, general anesthesia and endotracheal intubation of the patient. Hypopharyngeal burns of third degree are an absolute contraindication for esophagogastroduodenoscopy. The most severe complication is iatrogenic perforation, which fortunately is a very rare occurrence, however when esophagogastroduodenoscopy is performed one has always to take precaution. Introduction of flexible endoscopic tubes has made this invasive method safer. Esophagogastroduodenoscopy gives us useful data on the existence of post-corrosive injuries and if they are documented, then an adequate treatment has to be initiated as soon as possible (35).

Endoscopic classification of post-corrosive injuries in the upper gastrointestinal tract is of enormous importance in diagnosis and treatment of acute corrosive intoxications. Kikendall (36) suggested a classification in four grades (Table 1).

GRADE I:	edema and erythema of the mucosa
GRADE II A:	hemorrhage, erosions, blisters, superficial ulcers
GRADE III B:	circumferential lesions
GRADE III:	deep grey or brownish-black ulcers
GRADE IV:	perforation

Table 1. Kikendall's classification

Some authors use the classification by Zargar (37) (Table 2).

GRADE 0:	normal mucosa
GRADE I:	edema and erythema of the mucosa
GRADE II A:	hemorrhage, erosions, blisters, superficial ulcers
GRADE II B:	circumferential lesions
GRADE III A:	focal deep gray or brownish-black ulcers
GRADE III B:	extensive deep gray or brownish-black ulcers
GRADE IV:	perforation

Table 2. Zargar's classification

Endoscopic ultrasound and computerized tomography are useful procedures in diagnosis that can more precisely determine the depth of the corrosive injuries.

6. TREATMENT

The aim of the therapy is to prevent perforation and to avoid progressive fibrosis and stenosis of the esophagus and stomach. Possible perforation of the esophagus or stomach can be treated only surgically.

Emergency surgery – Emergency surgical intervention is indicated in cases of esophageal or gastric perforation although it is difficult to predict it initially. Patients with shock, coagulation disorders or acidosis and those who have ingested a large quantity of corrosive substances tend to develop severe post-corrosive injuries and laparotomy and resection of damaged segments may be beneficial in the treatment of these patients. Total esophagotomy or gastrectomy and placement of gastrostoma or jejunostoma for artificial nutrition are made. In one of his studies Zargar suggests prompt surgical intervention in patients with severe post-corrosive injuries (grade IIIB), thus advocating decrease of mortality and morbidity in these patients. Other authors are against urgent resection of the esophagus and stomach, explaining that the grade of the post-corrosive injuries cannot be always precisely determined (38, 39).

Neutralization of corrosive substances – A large number of authors think that neutralization is contraindicated because, in order to be effective, it must be done within the first hour after ingestion of a caustic agent. Alkalis can be neutralized with mild vinegar, lemon or orange juice. Acids can be neutralized with milk, eggs or antacids. Sodium bicarbonate is not recommended because it produces carbon dioxide, which increases the risk of perforation. Some authors think that the heat produced in the neutralization reaction increases the possibility of additional injuries of upper gastrointestinal tract. Emetics are contraindicated because of re-exposition to the corrosive substance leading to injury exacerbation. Activated charcoal is also contraindicated (40).

Antibiotics – The use of antibiotics in acute caustic poisonings is still under debate. Since there is not a sufficient number of controlled studies that would confirm the need of antibiotic use, many authors do not recommend them in treatment of caustic intoxication. However, controlled trials in animals have revealed that bacterial invasion of post-corrosively damaged mucosa and severe inflammation induce tissue granulation with a resultant formation of tissue fibrosis. That is why some authors justify the administration of broad spectrum of antibiotics, most commonly of the penicillin group (41).

Prevention of strictures – Stricture formation is the most severe late post-corrosive complication. Earlier studies have indicated that stricture formation can be prevented or reduced by steroid use, stenting, use of nasogastric tube and balanced diet, and retrograde intraluminal dilatation.

Corticosteroids – The use of corticosteroids in acute caustic poisonings continues to be a debatable issue. Several studies including 361 patients showed 19% of esophageal and stomach stenosis in patients treated with corticosteroids and 41% of stenosis in those not receiving corticotherapy. Dexamethasone of 1 mg/kg/day or prednisolone of 2 mg/kg/day was given to these patients (42).

Some studies did not prove the preventive effect of corticosteroids in stricture formation but have shown an increased risk of onset of peritonitis or mediastinitis. Such multicentric study comprising 572 patients conducted at the same time in several European countries indicated that corticosteroids have no significant influence on prevention of post-corrosive stenosis in acute corrosive poisonings (43).

Nutrition – Extensive damage of the gastrointestinal tract hinder physiological nutrition in these patients. Within a short period of time, these patients fell into a severe general condition due to hypercatabolic state and negative alkali balance (5). The type of the artificial nutrition depends on the degree of esophageal or gastric damage seen by endoscopy.

In patients with I and II A grade of damage, total

parenteral nutrition in the first 24-48 hours is followed by liquid diet until the 10th day. Afterwards, food intake can be in a more liberal regimen.

In patients with II B and III grade of damage the so-called "esophageal rest" is recommended, that is, the patient must not take food per os (NPO). During the "rest", the patient is fed by nasogastric or nasoenteral tube, gastrostoma or jejunostoma and parenterally by peripheral or central vein. Esophageal rest is explained with the fact that food particles enter the granulocytes of the esophageal wall and exacerbate the inflammation (16). Esophageal rest may last until the 10th day after corrosive ingestion or some authors say until the 15th day, that is, until the first endoscopic control. Intensive hyperalimentation and esophageal rest may reduce the percentage of late post-corrosive complications (stenosis and strictures), although this has not been clearly supported in controlled trials (44, 45).

Some authors recommend taking liquids (liquid nutritional solutions, milk) 48 hours post- ingestion if the patient can swallow his/her saliva (Figure 4).

Esophageal dilation – Retrograde intraluminal esophageal dilation is performed for prevention or dilation of the already created esophageal narrowing. According to some authors, it can be done immediately after injury or 15 days after ingestion. The predominant attitude is that it is safest to start with the esophageal dilation 6 weeks after ingestion. Then it is performed every 2 to 3 months in several consecutive time intervals (46).

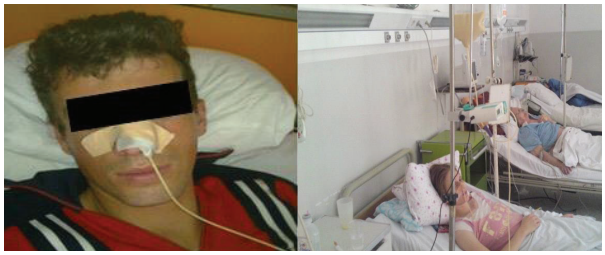

Figure 4. Our patients with a nasojejunal tube, who were fed by Eneroport pump

In spite of the presented positive experiences, this method is not recommended by many authors because it can traumatize the esophagus, can cause bleeding and esophageal perforation and can increase predisposition to fibrosis formation (Figure 5).

Sucralfate use – There are subjective reports that the use of sucralfate may decrease the percentage of post-corrosive stenosis in the upper gastrointestinal tract (47).

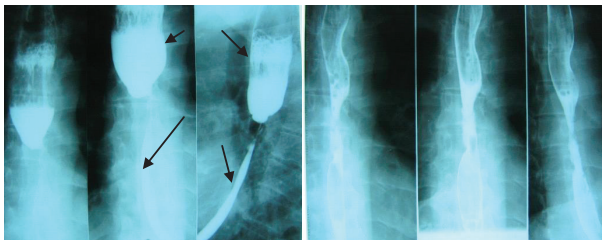

Figure 5. X-ray finding of narrowed mid- and distal esophagus and dilated proximal esophagus and X-ray finding of esophagus after retrograde intraluminal dilation

Esophageal stent – In patients with IIB and III grade of esophageal injuries a specially designed intraluminal stent under endoscopic guidance may be placed preventively or after onset of stenosis. The limited number of controlled studies has not given substantial support to this method (48).

Surgery – Surgical intervention is indicated when there is a:

- complete stenosis that cannot be treated with usual conservative methods;
- defect of the esophagus or stomach detected with x-ray examination;
- fistula formation.

Currently, the stomach, jejunum and colon are the most commonly used organs to replace the esophagus. Esophagectomy with colon interposition is the most frequently applied method in serious esophageal lesions (49). Gastric transposition is more recommended in children although there are data on high mortality rate (5%), anastomotic leakage (12%) 1and postoperative dilation due to onset of strictures (20%) (50).

Prognosis – Prognosis in acute corrosive poisonings is variable and depends on the degree of esophageal and gastric injury as well as on the general health condition of the patient. The highest mortality rate has been recorded as a result of perforation and mediastinitis.

6.1. Expert commentary

Acute corrosive poisonings are a serious social-medical issue, both from the sense of clinical presentation and the therapeutical approach as well.

Such poisonings cause severe chemical injuries of the upper gastrointestinal tract, most commonly localized to the esophagus and the stomach, presented as difficult clinical signs, in which case the clinical investigations are hard to perform, so the treatment and the outcome are often uncertain.

They are most commonly seen in the population at there most productive period of life and they present as an economical burden due to expensive diagnostic and therapeutical programs, and extended hospitalization.

The care for these cases involves a multidisciplinary approach, due to the difficult clinical presentation in the acute period, and the need for extended evaluation, severe post-corrosive complications and a possibility for a permanent disability.

6.2. Five-year view

In the next five years, there will be a need for controlled clinical studies, which will look for a new ways of reducing the high percent of post-corrosive stenosis of the upper gastrointestinal tract. There is a need to combine the alternate viewes of therapeutical approaches in acute corrosive poisonings, especially for the use of: antibiotics, corticosteroids, high dosage of antisecretory and antacid drugs, citostatics, perfecting of the modern methods of artificial nutrition.

6.3.Key issues

- In the future corrosive poisonings will remain a serious social-medical issue, due to the difficult clinical presentation, expensive diagnostic protocol, extended hospitalization and possible permanent disability.
- The urgent esophagogastroduodenoscopy remains the most valued standard tool in diagnosis and prognosis of the acute corrosive poisonings.
- The presence or absence of subjective difficulties and symptoms, local and objective clinical signs, can not be a conformation of presence or absence of the poisoning or eventual post-corrosive lesions.
- The use of corticosteroids is controversial as therapy. Some authors are in a conflict of using antibiotics and even antisecretory drugs.
- Of great importance, during the treatment, are the appropriate nutrition of the patients and maintaining of their nutritional status.
- Post-corrosive stenosis are the most common latent complication, and they still remain a high percentage.
- As a less common complication, carcinoma of the upper gastrointestinal tract may appear.
- An urgent surgical treatment is needed in cases with perforations during the acute or chronic period of the poisoning.

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