

POST-CORROSIVE INJURIES OF UPPER GASTROINTESTINAL TRACT

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Abstract: Acute poisonings with corrosive substances may cause serious chemical injuries to upper gastrointestinal tract, the most common location being the esophagus and the stomach. If the patient survives the acute phase of the poisoning, regenerative response may result in esophageal and/or gastric stenosis and increased risk for esophageal cancer. 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.

In establishing the diagnosis of acute corrosive poisonings, the severity of the post-corrosive endoscopic changes of the esophagus, stomach and duodenum is of major importance. According to Holinder and Fridman classification, post-corrosive endoscopic changes are classified in three degrees:

First degree – superficial damage associated with hyperthermia, epithelial desquamation and mucous edema.

Second degree – transmucous damage affecting all of the mucosal layers, followed by exudation, erosions and ulcerations.

Third degree – transmural damage associated with ulcers' penetration in the deep layers of the tissue and neighboring organs.

Severity of the lesions depends on the nature, quantity and concentration of the corrosive substance, the duration of exposure and current state of the exposed organs.

Most often caustic injuries occur to the esophagus and stomach since the corrosive substance remains there for a longer period of time. Treatment of the acute corrosive intoxications include: neutralization of corrosive agents, antibiotics, corticosteroids, anti-secretory therapy, nutritional support, collagen synthesis inhibitors, esophageal dilation and stent placement, and surgery. The most common complications that

may appear are: perforation, gastrointestinal bleeding, sepsis, esophageal strictures and stenosis, stenosis of gastric antrum and pylorus, cancer of the esophagus and the stomach.

Today, owing to the substantially enhanced diagnostic and therapeutic approach, the mortality percentage has been reduced from 20% to 1–5%. Women more often than men are intoxicated with corrosive substances; suicidal poisonings prevail; the most abused agents are hydrochloric acid (HCl) and sodium hydroxide; intoxications are more common in children (80% out of the total number of intoxications).

In spite of the preventive measures for restriction of the trade with corrosive substances, standardization of their concentration and protective safety bottle caps, still the number of corrosive intoxications, the percentage of post-corrosive complications and the handicap are high. Acute corrosive intoxications are the leading cause of death in clinical toxicology.

Key words: corrosive intoxications, esophageal burns, corrosive substances, esophago-gastroduodenoscopy.

Introduction

Acute corrosive poisonings appear as a result of ingestion of acids, bases, oxidants, heavy metal salts and other chemical substances. Modern technology has enabled synthesis of chemical substances with strong corrosive features that are often used in everyday life [1].

Injuries caused by poisonings with corrosive agents are as old as are the corrosive chemicals. From ancient times corrosive alkalis were mixed with oil, and soap was prepared; from vapors of concentrated solution of ashes, a mixture of potassium and sodium carbonate was created with corrosive features. Caustication of this mixture is one of the oldest chemical-technological procedures for production of corrosive substances [2].

Modern technology enables synthesis of corrosive agents that are widely applied in the industry, households and everyday life.

The synthesis, production and usage of corrosive agents with high concentration and different Ph value (Ph greater) have increased the risk for abuse and poisoning.

The route of entry of corrosive substances in the body is:

- oral (ingestion of a corrosive agent), and rarely
- inhalation (inhalation of corrosive vapors) [3].

Contrary to past indicators, today purity and concentration of the corrosive substances is higher causing more severe forms of poisonings. The severity of the chemical burns that appear and affect almost the entire gastrointestinal tract depends on several factors:

- nature of the corrosive substance;
- quantity ingested;

- its concentration;
- duration of exposure;
- the act of swallowing;
- the existing condition of the tissues that came in contact with the corrosive agent.

Therefore, poisonings present with severe clinical signs; clinical investigation is difficult and therapy and outcome unpredictable [4].

Corrosive substances

Oral intoxication with corrosive agents is caused by ingestion of:

- acids (hydrochloric, acetic, sulfuric, oxalic, carbolic),
- alkalis (sodium and potassium, soaps, detergents),
- heavy metal salts (sublimite),
- formalin,
- iodine tincture and many other chemical substances [5, 6, 7].

One broad term "lye" is found in the American literature data, which implies strong alkalis used in the cleansing agents [8, 9, 10, 11].

In our country the most common abused acid is hydrochloric acid (HCl), which is easily accessible as a sanitary cleansing agent. Intoxication, although rarely, is caused by antirust compounds (oxalic acid), by sodium hypochlorite (NaClO₄), which as a solution or combined with hydrochloric acid is used for cleaning sanitary facilities and swimming pools, as a chemical agent in batteries fluid (sulfuric acid – H₂SO₄), household bleaches (5% of Na hydrochloride) [12, 13]. Rarely observed are also acute intoxications with acetic acid (CH₃COOH), which is used in the food industry for vegetables conservation and is often abused during the season of preparing food for winter, as well as hydrogen peroxide that is used as a cosmetic agent in a concentrated form [14].

Beside acids, corrosive alkalis are also being abused, such as sodium hydroxide (NaOH) and potassium hydroxide (KOH). They are found on the trade market in paste or granular forms. They have Ph value greater than or equal to 12. They are also 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 [15, 16, 17].

In an earlier study conducted at our Clinic, 37 patients with acute caustic poisonings were examined and treated. Of them, 28 (75.7%) patients ingested hydrochloric acid, 6 (16.2%) acetic acid and 3 (8.1%) sodium hydroxide [15]. In another study, we treated 86 patients with acute caustic poisonings, of whom 72.2% or 62 patients ingested acids and 27.9% or 24 patients ingested alkalis [1].

Pathophysiology and pathology

Solutions with Ph smaller than 2 or greater than 12 are highly corrosive and can cause severe chemical burns in the upper gastrointestinal tract. The most serious lesions occur in the esophagus and the stomach since the poison remains there a long time [18, 19, 20, 21]. Corrosive damages caused by oral ingestion of corrosive substances are a result of the tissue colloid condition. The route of the corrosive agent through the gastrointestinal tract causes extensive damages in the oral cavity, throat, esophagus, stomach, and duodenum [22].

A concentration of 22,5% NaOH solution can produce perforation of the esophagus and/or stomach in 10 seconds.

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 proteinate and fats into soaps, resulting in penetrating or liquefaction necrosis [23].

Few hours after ingestion of a corrosive substance, thrombosis of the small vessels appears producing heat that initiates 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 [12]. Consequently, collagen deposition is minor until the second week after ingestion, and the healing process begins three weeks after ingestion [24]. Because of these changes, some authors do not recommend esophagogastroduodenoscopy between the 5th and the 15th day after corrosive ingestion [25]. Three weeks after ingestion, tissue fibrosis occurs, resulting in narrowing of esophageal and/or stomach lumen and onset of strictures [26] (Table 1).

Table 1 – Табела 1

*Algorithm of post-corrosive damage to the wall
of the injured organ (esophagus – stomach)
Алгоритам на посткорозивније промени во судоџ
на оштееениој орган (езофаџус – желудник)*

Timing of Tissue Damage and Repair	
Injury	Time
Acute injury	Day 0
Inflammation, vascular thrombosis	1 to 7 days
Granulation tissue	10 to 21 days
Fibrosis/stricture	3 weeks

Caustic ingestion: Pathophysiology, diagnosis, management. Ferry GD, 1993.

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 wall of the esophagus and stomach. 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 develops at the cricopharyngeal area, at the level of the aortic arch and tracheal bifurcation and the lower esophageal sphincter. Most gastric stenoses occur in the atrium and pylorus [27].

The third degree is characterized by perforation of the wall of the esophagus or stomach.

Clinical features

Clinical presentation of corrosive injuries in the upper gastrointestinal tract depends on the physical state, type and quantity of the corrosive substance. Corrosive agents in powder or crystal state adhere to oral cavity and throat, causing the most severe injuries to these organs as opposed to the liquid agents that pass rapidly through the esophagus and cause severe corrosive burns to entire organ with predilection of the cricopharyngeal area, at the level of the aortic arch and the lower esophageal sphincter [28]. The most severe gastric injuries are those of the antrum and pylorus where the caustic substance remains for a very long time [29]. Absence of changes in the oropharynx does not exclude severe injuries of the other areas of the gastrointestinal tract. Hypersalivation, difficulty in swallowing, edema, ulceration or whitish plaques in the oral cavity, palatal mucosa and pharynx are common phenomena [30, 31, 32]. Absence of oropharyngeal local changes does not exclude severe esophageal injuries. In 10% to 30% of the 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, oropharyngeal injuries are not a reliable indicator for the eventual damages of the esophagus [35, 36, 37] (Figure 1.)

Local obvious lesions are painful and contact bleeding. Hoarseness induces laryngeal, epiglottal or hypopharyngeal complication. High temperature accompanied with fever induces perforation and suggests consultation with a surgeon [11]. 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 may be delayed for few hours after ingestion and they may last days and weeks [2]. 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 and bronchial necrosis with mediastinitis, leading to death [38].



Figure 1 – Patient with severe post-corrosive injuries

Слика 1 – Пацієнтка со йеишки локални йосйкорозивни йромени

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. This requires emergency surgery. These injuries may appear in the first 48 hours or they may be delayed until the 14th day after corrosive ingestion [39].

The most usual sites of corrosive lesions in the upper gastrointestinal tract are presented in Table 2.

Table 2 – Табела 2

The most common location of the corrosive injuries
Најчешћа локализација на корозивни повреди

Pharynx	10%
Esophagus	70%
Upper segment	15%
Mid segment	65%
Lower segment	20%
Entire organ	18%
Stomach	20%
Antrum	91%
Whole stomach	90%

Late complications

Late complications are a major problem in acute corrosive poisoning and often cause permanent handicap in patients. Sophisticated diagnostics and treatment over the last several years have substantially reduced the percentage of late complications related to acute corrosive intoxications and injuries in the upper gastrointestinal tract [40, 41, 42]. 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 pylorus, esophageal and stomach cancer [43].

Strictures and stenosis of the esophagus – may appear three weeks after ingestion of the corrosive substance, in the first three months or even after one year according to some authors. Liquid corrosive substance ingestion more often initiates stenosis than corrosive substances in crystal state [44] (Figure 2).

Stenosis of antrum and pylorus – feeling of full stomach, nausea, vomiting, 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 [45, 46] (Figure 3).

Esophageal and stomach cancer – the latent period from the ingestion of the corrosive substance and the development of cancer may range between 40 to 50 years. According to some studies, 3% of esophageal cancers present with a

history of previous caustic substance ingestion. The most common location is the level of carina and prognosis of these esophageal cancers is better than esophageal cancers of other etiology. Gastric cancer is a very rare complication following corrosive ingestion [22].

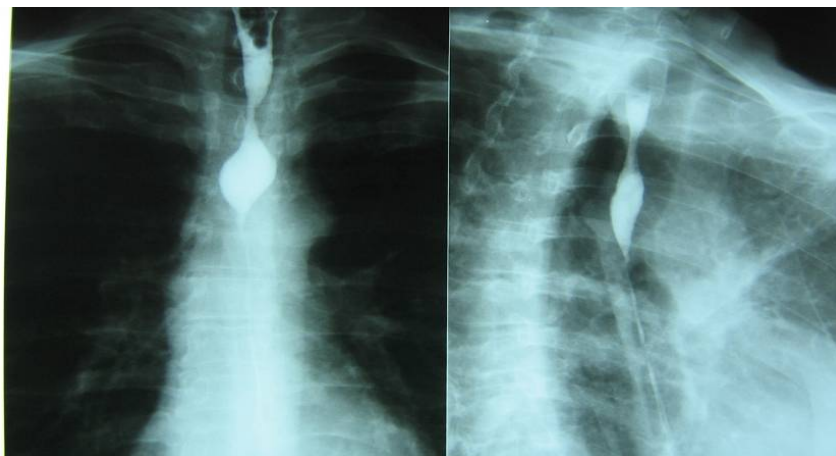


Figure 2 – Post-corrosive narrowing of the mid- and distal esophagus
Слика 2 – Посйкорозивно сйеснување на среднои и дисйален дел на хранојпроводникои

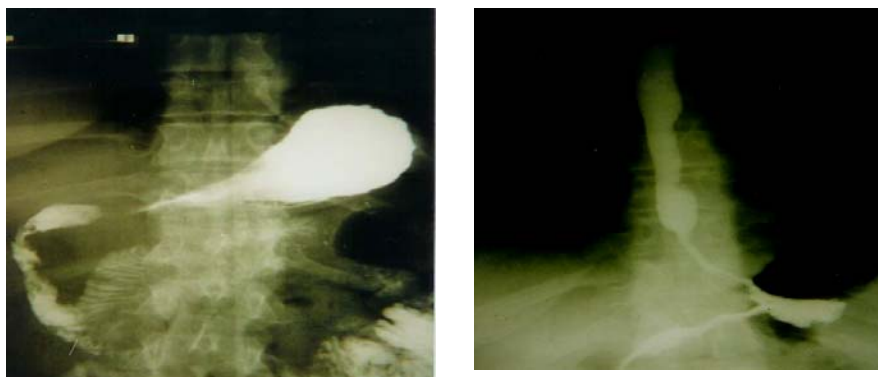


Figure 3 – X-ray finding of stenosis of gastric antrum and pylorus
Слика 3 – РТГ наоди со сйеноза на анйрален и пилорен дел на желудник

A study comprising 86 patients showed that 25–30 days after corrosive ingestion, 27.8% of the patients developed stenosis presented on the first endo-

scopic and x-ray control. Of the 86 patients, 25.8% were intoxicated with acids and 66.6% with alkalis. The most common location of the stenosis was the distal part of the esophagus and gastric antrum and pylorus with 6.4% in both cases. In 16.7% of the patients who ingested alkalis, stenosis developed in the middle and in the distal part of the esophagus [15].

In another study, 37 patients with acute corrosive poisoning were monitored for six months. After the conducted therapy, 28 of them (75.7%) had a normal finding, 7 (18.9%) had superficial changes associated with hyperemia and edema and 2 (5.4%) had stenosis on the distal part of the esophagus. Endoscopic and x-ray examination of the stomach revealed normal finding in 16 (43.2%) of the patients, hyperemia and edema in 11 (29.7%), stenosis of antrum, corpus and pylorus in 7 (18.9%), stenosis of antrum and corpus in 2 (5.4%) and stenosis of pylorus in 1 (2.7%) patients (1).

Management

Radiologic studies – in the acute phase of caustic injury, plain chest and abdominal roentgenograms may reveal presence of mediastinal air or free air under the diaphragm that may be the evidence of esophageal or gastric perforation [47].

Esophagogastroduodenography with gastrographin 25–30 days after corrosive ingestion may give us useful information on changes in the dimensions of esophageal and gastric lumen [48]. Some authors prefer barium sulphate as less irritant, especially in case of its aspiration and to be used immediately after ingestion for monitoring the development of complications [49].

Esophagogastroduodenoscopy – a sophisticated and sovereign method for diagnostic evaluation of acute corrosive intoxications and lesions of the upper gastrointestinal tract [50]. According to the latest controlled studies the most optimal timing for esogastroduodenoscopy is the first 12–24 hours after corrosive ingestion [5]. (Figure 4)

Some authors suggest that upper endoscopy may be safely performed at least in the first 96 hours after the caustic ingestion. Since the healing process of the post-corrosive injuries begins the 4th day and is most intensive until the 14th day, it is suggested to avoid this diagnostic procedure [18]. Making a decision on emergency esogastroduodenoscopy depends on several factors: type of the corrosive substance, its quantity, the intention of ingestion the corrosive substance and onset of symptoms following the ingestion.

The latest researches suggest an eventual emergency upper endoscopy to be carried out after previous sedation, general anesthesia and endotracheal intubation of the patient [49].

Hypopharyngeal burns of third degree are an absolute contraindication for esophagogastroduodenoscopy [50]. The most severe complication is iatrogenic perforation, which fortunately is very rare, however when done one has always to take precaution.

Esophagogastroduodenoscopy gives 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 [38].

Endoscopic classification of post-corrosive injuries in the upper gastrointestinal tract is of enormous importance in diagnosis and treatment of acute corrosive intoxications. Kikendall [31] suggested a classification in four grades:

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

Some authors use the classification by Zargar:

- 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 [18].

Endoscopic ultrasound and computerized tomography can be used in order to determine more precisely the depth of the corrosive injuries [11].

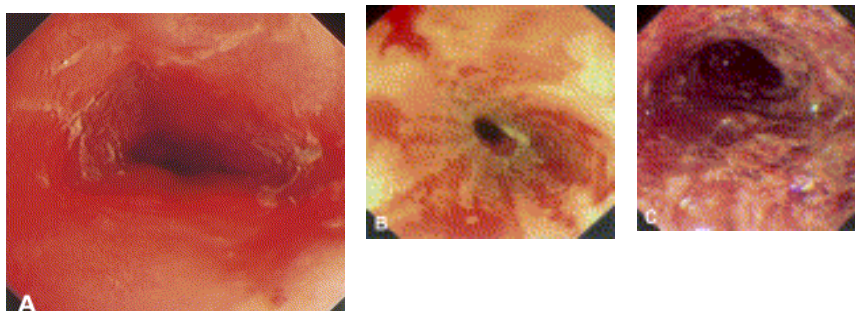


Figure 4 – Endoscopic finding of severe post-corrosive injuries
Слика 4 – Ендоскојски наод за њежки њосћќорозивни њовреди

Gastric lavage, induced vomiting and ingesting of active coal are contraindicated because of the possibility of esophageal re-exposition to corrosive substance, producing additional injuries [51]. Some authors suggest the use of milk and water as an antidote, however, their efficacy has not been confirmed by many controlled studies. Milk may compromise the urgent esophagogastro-duodenoscopy [25].

Treatment

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

Emergency surgical intervention is indicated in cases of esophageal or gastric perforation although it is difficult to predict it initially. Emergency surgery is the unique choice in these situations [11]. Some authors recommend emergency surgery in patients with severe post-corrosive injuries of the esophagus and stomach of III grade [13]. Total esophagotomy or gastrectomy and installation of gastrostoma or jejunostoma for artificial nutrition are made [52].

Neutralization of corrosive substances: A large number of authors who are involved with the problem of caustic intoxications think that neutralization is contraindicated. In order to be effective, it must be done within the first hour after ingestion of a caustic agent [31]. Alkalis can be neutralized with mild vinegar, lemon or orange juice. Acids can be neutralized with milk, eggs or antacids [12]. 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 [44].

Emetics are contraindicated because of re-exposition to corrosive substance leading to injury exacerbation. Active coal is also contraindicated.

Corticosteroids – 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 [53, 54]. Some studies did not prove the preventive effect of corticosteroids in stricture formation but corticosteroids may increase the 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 had no

significant influence on prevention of post-corrosive stenosis in acute corrosive poisonings [55, 56].

Antibiotics – Controlled studies performed in animals 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 [57]. Since there is no sufficient number of controlled studies that would undoubtedly confirm the need of antibiotic administration, many authors do not recommend them in the treatment of caustic intoxications [22].

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. Nutrition is life-maintaining therapy in patients who cannot take food and are disposed to a risk of malnutrition [5]. Documented effects from artificial feeding in patients intoxicated with corrosive substances are:

- reduction of infections,
- reduction of predisposition to developing aspiration pneumonia,
- reduction of the risk for pulmonary embolism,
- economical reasons.

The type of the artificial nutrition depends on the degree of esophageal or gastric damage seen by endoscopy [12].

In patients with I and II A degree of damage, total parenteral nutrition in the first 24–48 hours is followed by a liquid diet until the 10th day. Afterwards, food intake can be in a more liberal regimen.

In patients with II B and III degree of damage the so-called “esophageal rest” is recommended, that is the patient must not take food per os. During the “rest”, the patient is fed by nasogastric or nasoenteral tube, gastrostoma or jejunostoma and parenterally by peripheral or central vein. This is explained by the fact that food particles enter the granulocytes of the esophageal wall and exacerbate the inflammation [16] (Figures 5, 6, 7). 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 [58]. Some authors recommend taking liquids (liquid nutritional solutions, milk) 48 hours after ingestion if the patient can swallow his/her saliva [50].



Figure 5 – Patient with a nasogastric feeding tube
Слика 5 – Пациентка со инсталирана насојејунална сонда



Figure 6 – Patient with nasogastric tube on enteral nutrition through enteral pump
Слика 6 – Пациентка со насојејунална сонда на ентерална нутриција преку ентеројорџи пумпа



Figure 7 – Installed isolated gastrotomy for decompression and feeding jejunostoma
Слика 7 – Приказ на инсталирана изолирана гастријосијома за декомпресија и Feeding јејуносијома

Esophageal dilation – intraluminal esophageal dilation is performed for prevention or dilation of the already created esophageal narrowing [59]. 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 in every 2 to 3 months in several consecutive time intervals [60] (Figure 8, 9).

In spite of the presented positive experiences, this method is not recommended by many researches because dilation can traumatize the esophagus, bleeding and esophageal perforation can occur and increased predisposition to fibrosis formation [61].

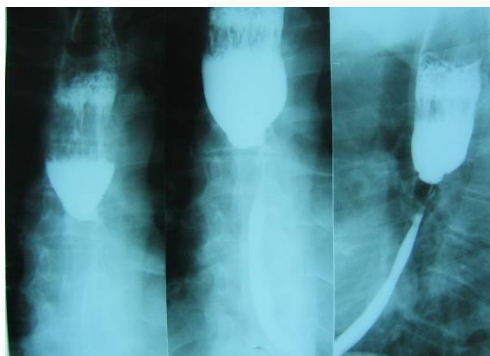


Figure 8 – X-ray finding of narrowed mid- and distal esophagus and dilated proximal esophagus

Слика 8 – РТГ наод за ситеснеи среден и дисџален дел и дилајџиран џроксимален дел на хранојровод

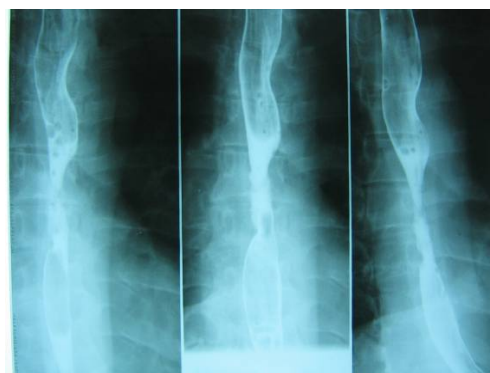


Figure 9 – X-ray finding of an esophagus after retrograde intraluminal dilation

Слика 9 – РТГ наод на езофаџус џо рејџроџраднa инџралуминална дилајџација

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 insufficient number of controlled studies has not given substantial support to this method.

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;
- development of a severe periesophageal reaction with mediastinitis

and dilation;

- fistula formation [61].

Currently, the stomach, jejunum and colon are the most common organs used to replace the esophagus. Esophagectomy with colon interposition is the most frequently used method in serious esophageal lesions [42]. Gastric transposition is more recommended in children although there are data on the high mortality rate (5%), anastomotic leakage (12%) and postoperative dilation due to onset of strictures (20%) [61].

Recommendations – In spite of the good screening results and modern treatment of patients with acute corrosive poisonings, the American Society of Gastrointestinal Endoscopy (ASGE) gives several recommendations for monitoring of patients with caustic injuries to upper gastrointestinal tract:

- surveillance of patients 15 to 20 years after corrosive ingestion;
- endoscopic examination every three years;
- reduction of the threshold for evaluation of dysphagia (11).

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.

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Резиме

ПОСТКОРОЗИВНИ ПОВРЕДИ НА ГОРНИОТ ДЕЛ НА ГАСТРОИНТЕСТИНАЛНИОТ ТРАКТ

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Акутните труења со корозивни супстанции можат да предизвикаат тешки хемиски повреди на горниот дел на гастроинтестиналниот тракт најчесто локализирани на езофагусот и желудникот. Ако пациентот ја преживее акутната фаза на труењето, регенеративниот одговор може да резултира со езофагеална и/или гастрична стеноза и зголемен ризик од карцином на езофагусот. Акутните корозивни труења претставуваат мајорен проблем во клиничката токсикологија, бидејќи најчесто е загрозувана популацијата на млади луѓе кои се психички растроени, склони кон самоубиство и алкохолизам.

При дијагнозата на акутните корозивни труења од голема важност е и тежината на постокорозивните ендоскопски промени во езофагусот, желудникот и дуоденумот. Според класификацијата на Holinger и Fridman посткорозивните ендоскопски промени се класифицирани во три степени:

Прв степен: суперфицијално оштетување проследено со хиперемија, десквамација на епителот и едем на слузницата.

Втор степен: трансмукозно оштетување проследено со зафаќање на сите слоеви на слузницата, ексудација, ерозии и улцерации.

Трет степен: трансмурално оштетување проследено со пенетрација на улцерациите во длабоките слоеви на ткивата и околината.

Тежината на лезијата зависи од природата, количината и концентрацијата на корозивниот агенс, времетраењето на експозицијата и моменталната состојба на органите кои се експонирани.

Најчесто повредите од ингестијата настануваат на езофагусот и желудникот, бидејќи во нив корозивот се задржува најдолго. Во третманот на акутните корозивни труења се вклучени: неутрализација на корозивниот агенс, антибиотици, кортикостероиди, антисекреторна терапија, нутритивна поддршка, колаген синтетички инхибитори, езофагеална дилатација и стентирање, хируршки третман. Најчести компликации кои можат да се појават се: перфорација, гастроинтестинално крвање, сепса, езофагеални стриктури и стенози, стеноза на антрум и пилорус на желудникот, карцином на езофагусот и желудникот.

Благодарение на значајно подобриот дијагностички и терапевтски пристап денес процентот на морталитет е намален од 20% на 1–5%. Поголемиот број на пациентите интоксичирани со корозиви се жени, доминираат суицидалните труења, најчесто злоупотребено средство е хлороводородната киселина (HCl) и натриум хидроксид (NaOH) и труењата се почести кај деца (80% од вкупниот број на труења).

И покрај сите превентивни мерки за ограничување на прометот на корозивни супстанции, стандардизирањето на нивната концентрација и заштитните сигурносни затвораи на шишињата, бројот на корозивни труења, процентот на посткорозивни компликации и инвалидитетот се високи. Акутните корозивни труења се водечка причина за смртен исход во клиничката токсикологија.

Клучни зборови: корозивни труења, езофагеални изгореници, корозивни супстанции, езофагогастроуденоскопија.

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