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EPIDEMIOLOGY OF SEVERE POISONINGS CAUSED BY INGESTION OF CAUSTIC SUBSTANCES

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Abstract: Acute poisoning with corrosive substances can cause severe chemical injuries of the upper part of the gastrointestinal tract. They are most frequently localized in the oesophagus and stomach. If the patient survives the acute phase of poisoning, the regenerative response can result in oesophageal and/or gastric stenosis and a higher risk of oesophageal and stomach cancer.

Seven years' clinical material was evaluated for this study (2000–2006) with a total number of 517 patients, hospitalized and treated at the Urgent Internal Medicine and Toxicology Clinic, University Clinical Centre, Skopje, Republic of Macedonia.

The evaluation of the caustic poisonings was followed on the basis of anamnesis and hetero-anamnesis, physical examination of the patient, and status of the local changes to the oral mucosae, tongue, palate, and pharynx.

The patients were assessed by the following techniques of visualization: urgent oesophagoduodenoscopy in the first 24 hours after the ingestion, control oesophagoduodenoscopy (15 and 25 days after ingestion), X-ray of oesophagus, gaster and duodenum with gastrograph (25–30 days after the ingestion), as well as a routine laboratory examination (blood count, urea, creatinine, enzyme, protein and lipid status, serum transferin, etc), following also the actual body mass index (BMI).

The presented results show that the dominating patients were female [n = 368 patients (71.79 %), $p < 0.001$] and Christian by religion [n = 263 (50.87 %), $p > 0.05$], with the majority having had secondary education [n = 322 (62.28 %), p = 0.001].

The most frequently misused substance was chlorine hydrogen acid [n = 245 patients (47.38%)]., At the first urgent oesophagoduodenoscopy examination the majority of patients had II A (n = 190) and II B (n = 136) grade damage (x^2 = 44.0; $p < 0.05$). One hundred patients, or 19.34 %, were dismissed from the hospital with stenosis of the oesophagus and stomach. The high percentage of stenosis was the major factor in the invalidity of these patients. A high mortality of 8.69 % ($p > 0.05$) was also stated.

Of 517 patients, 62 (11.99 %) were poisoned with concentrated acetic acid. In 37 patients (59.67 %) acute renal failure developed as an acute complication and four patients (6.5 %) died as a consequence of the disordered renal function and the need for active dialysis treatment. Recovery of the renal function was established in 93.5 % of the patients. A total number of 138 haemodialyses (3.7/patients) were performed.

Acute corrosive poisonings represent a serious socio-economic problem, as well as a diagnostic and therapeutic problem. They appear most frequently in a population that is in a period of life when the person is most creative and efficient in terms of working capacity. The treatment is expensive and is an economic burden on the social community.

Despite all the current therapeutic treatments and efforts made to decrease the mortality and late morbidity, intoxication with corrosive substances remains a difficult medical problem.

Key words: caustic poisonings, oesophagoduodenoscopy, oesophageal and stomach corrosive injuries.

Introduction

Acute corrosive poisonings are the consequence of an ingestion of acids, bases, oxidants, heavy metal salts and other chemical substances. Poisonings are presented with serious clinical manifestations, clinical investigations are frequently impeded and the therapy and the course of the disease are often uncertain.

The ingestion of corrosive substances leads to severe damages in the upper part of the gastrointestinal tract (GIT). The severity of these lesions depends on the nature of the corrosive substance, its quantity and concentration, and the duration of exposure.

In the diagnosis and treatment of corrosive poisoning the severity of post-corrosive endoscopic changes in the oesophagus, stomach and duodenum is of great importance. According to the Holinger and Friedman classification, post-corrosive endoscopic changes are classified on three levels: 1) superficial damage followed by hyperaemia, desquamation of the epithelium and oedema of the mucous membrane; 2) trans-mucous damage followed by inclusion of all mucous layers, exudation, erosions and ulceration; 3) trans-mural damage

followed by penetration of ulceration into the deep-tissue layers and surroundings.

The most severe damage appears in the oesophagus and stomach because the agent is kept there for longer period of time. Hence, the most frequent localization of the late complication (stenosis) is at the level where the physiological oesophagus narrows or the stomach.

The aim of the therapy is to prevent perforation and to disable progressive fibrosis and stenosis of the oesophagus and stomach.

Gastric lavage indicated by vomiting and administration of active coal are contraindicated because of the possibility of re-exposure of the oesophagus to the corrosive agent, causing an additional injury. Anti-secretion therapy, antibiotics, cortico-steroids, nutritive support, oesophageal dilatation and stenting, as well as surgical treatment, are well-established therapeutic approaches.

The aim of our study was to give an epidemiologic survey of caustic poisonings in the clinical toxicology, to evaluate the severity and significance of the late post-corrosive complications, invalidity and cost to the social community with respect to investigation and corresponding treatment.

Material and methods

Clinical material from patients hospitalised and treated at the Urgent Internal Medicine and Toxicology Clinic in the period from 2000 to 2006 was analysed. The patients were aged 15 to 75. The evaluation of caustic poisonings was followed on the base of anamnesis and hetero-anamnesis, physical examination of the patient and status of the local changes in the oral mucosae, tongue, palates, and pharynx. The following demographic and clinical data were analysed: sex, religion, degree of education, type of corrosive substance ingested during poisoning, the motive for poisoning, actual body mass index (BMI), the first urgent endoscopic finding, and the endoscopic finding on discharge from the hospital.

The following techniques of visualisation were used: urgent oesophagoduodenoscopy in the first 24 hours following ingestion, control oesophagoduodenoscopy 15 and 25 days after ingestion, and X-ray of the oesophagus, gaster and duodenum with gastrograph 25–30 days after ingestion. The biochemical parameters followed as a part of the routine daily practice were: blood count, urea, creatinine, enzyme, protein and lipid status and serum transferin.

Mortality analysis was made, and special attention was paid to patients who were transferred to the Surgical Clinics for further treatment because of severe injuries and life-threatening complications. In addition, a group of

patients intoxicated by concentrated acetic acid was analysed separately because of the severe haemolysis and ensuing acute renal failure (ARF) as a severe, sometimes life-threatening acute complication. Of the total number of acute poisonings with concentrated acetic acid (62), as many as 37 developed the condition of ARF in the first 48 hours after poisoning, and were subsequently treated by haemodialysis.

The comparison between the groups for the numerical parameters was made by Student's t-test, while chi square statistical analysis was used as a method for testing the categorical variables. A P value less than 0.05 ($p < 0.05$) was considered to be statistically significant. The study analysis data were made using the Statistica statistical programme, release 7.0.

Results

At the Urgent Internal Medicine and Toxicology Clinic 3,269 patients with different types of poisonings were hospitalized and treated in the period from 2000 to 2006. Of them 517 or 15.81% were poisoned by a corrosive substance. Because of early, severe and life-threatening post-corrosive complications 15 patients or 2.90% were transferred for further treatment at the Surgical Clinics.

Demographic data, the motive for poisoning and the type of corrosive substances are presented in Table 1. A significantly greater number of female patients was treated [n = 368 (71.2%), $p < 0.001$]. There was no significant difference concerning religion; 263 (50.9%) were Christian and 245 (47.4%) of the patients were Muslim. The majority of patients had high school education n = 322 (62.3%), n = 175 patients (33.8%) had primary education, and only 20 patients (3.9%) had university level education [$p < 0.001$]. In terms of the reason for poisoning, n = 490 (94.8%) patients ingested the corrosive with suicidal intentions, while n = 27 (5,2%) patients poisoned themselves accidentally [$p < 0.001$]. In terms of the caustic substances the greatest number of patients (n = 245) were intoxicated by chlorine hydrogen acid (48.1%) 114 (22.1%) by sodium chlorate, 82 (15.8%) by sodium hydroxide, n = 62 (12.0%) by concentrated acetic acid, 6 (1.2%) by sulphuric acid, and only 4 (0.8%) – patients were intoxicated by hydrogen peroxide.

In the group of patients [n = 62 (11.99%)] who were poisoned with concentrated acetic acid 138 haemodialyses were performed or 3.7 haemodialyses per patient because of the development of acute renal failure. In 25 patients (40.3%) the renal function was reestablished conservatively, 18 patients (29.0%) underwent 3 dialyses each, 11 patients (17.7%) needed 4 dialyses each, and in 8 patients (13.0%) up to 5 dialyses were performed until renal function

and normalisation of the degradation products and electrolyte status were established (Table 2). Because of the need for renal replacement therapy (dialysis treatment), which is considered to be an additional complication, 4 (6.5%) patients died while the renal function was reestablished in 58 (93.5%) of the patients after a few dialysis sessions.

Table 1 – Tabela 1

Clinical, data on the reason for poisoning and level of education of patients in the study (number and percentage)
Клинички податоци, мотив и ниво на едукација на пациентите во студијата (број и проценти)

Sex (n & %)	Men	Women	P – value
	149 (28.8)	368 (71.2)	< 0.05
Religion	Christian	Muslim	
	268 (51.9)	249 (48.1)	> 0.05
Motive	Suicide	Accident	
	490 (94.8)	27 (5.2)	< 0.05
Education	University	Secondary	Elementary
	20 (3.9)	322 (62.3)[a]	175 (33.8)[a, b]

[a]P < 0.01 vs. university education,
[a, b]P < 0.05 vs. secondary education

Table 2 – Tabela 2

Type of chemical substance by which patients in study poisoned themselves (number and percentage)
Тип на хемиска супстанца со која се затруени пациентите во студијата (број и проценти)

Chemical poisoning substance					
HCl	NaOH	CH_3COOH	H_2SO_4	$(NaClO_4)$	H_2O_2
249 (48.1)[a]	82 (15.8)	62 (12)	6 (1.2)	114 (22.1)[b]	4 (0.8)

[a]P < 0.01 vs. all others,
[b]P < 0.05 vs. CH_3COOH, H_2SO_4, H_2O_2

Urgent oesophagoduodenoscopy performed in the first 24 to 28 hours in all patients showed that the most of the cases were classified at II A degree [n = 186 (36.0%)] and II B [n = 132 (25.5%)], while 124 (23.2%) patients were registered with III degree. The first (I) degree was registered in only 79 patients or 15.3%. Although the predominant classification at present distribution is II A

injury degree ($p < 0.05$) and assumed to be an easier level of post-corrosive injury in comparison with II B and III, it is shown that in clinical toxicology there is always a need for a serious approach in diagnosis and treatment (Table 3).

Table 3 – Tabela 3

Data for the number of patients (62) and dialyses (138) stratified in groups of poisonings with acetic acid and development of ABI (number and percentage)
Податоци за бројот на пациенти (62) и дијализи (138) стратифицирани по групи на труења со оцетна киселина и развој на АБИ (број и проценти)

Patients (n & %)	Dialyses (n & %)	Lethal result (n & %)
25 (40.3)	0 (0)	/
18 (29.0)	3 (39.1)	/
11 (17.7)	4 (31.9)	/
8 (13.0)	5 (29.0)	4 (6.5)

After the given course of therapy and treatment, control oesophagoduodenoscopy was performed at 25–30 days after the ingestion. Here, the majority of patients had normal findings [n = 403 (80.7%)], 31 patients or 6.0% developed oesophagus stenosis, 42 or 8.1% had stenosis of the antrum pyloric part of the stomach, and 27 patients or 5.2% developed combined stenosis of the oesophagus and antrum pyloric part of the stomach. The total percentage of late complications was [n = 100 (19.3%)]. Hence, at the presented distribution, a predominant group of patients presented with normal findings at discharge from hospital ($p < 0.05$), which is the result of current diagnosis and treatment of these poisonings in our institution (Table 4).

Табле 4 ‡ Tabela 4

Data and findings of the urgent oesophagoduodenoscopy in the first 24–48 hours (number and percentage)
Податоци за наодите од ургентната езофагодуоденоскопија во првите 24–48 часа (број и проценти)

Finding at admission to hospital	(n & %)	Finding at discharge from hospital	(n & %)
I	79 (15.3)	Normal	417 (80.7)
II A	186 (36.0)	Stenosis oesophagei	31 (6.0)
II B	132 (25.5)	Stenosis antropylorica	42 (8.1)
III	120 (23.2)	Stenosis oesophagei et antropylori	27 (5.2)

Of the total number of patients, 29 (5.6%) died in the first 96 hours after hospitalization, and 16 patients (3.1%) died in the period from the 4th to the 30th day after hospitalization. Thus the overall mortality was 45 out of 517 patients (8.69%) (Table 5).

Table 5 ‡ Tabela 5

Outline of mortality (number and percentage) of all patients in first 4 days, following interval up to 30 days, and number of patients for urgent surgical treatment
Приказ на морталитетот (број и процент) во врска со првите 4 дена кај сите пациенти, следниот интервал до 30 дена и бројот на пациенти за ургентен хируршки третман

Total mortality	In the first 4 days (n & %)	From 4 – 30 days (n & %)	For surgery (n & %)
45	29 (5.6)	16 (3.1)	15 (2.9)

Discussion

Acute corrosive poisonings are a serious medical and social problem, both from the aspect of clinical presentation and from the aspect of therapeutic approach.

Nowadays, the incidence of acute corrosive poisonings is increasing constantly [2, 4, 6, 30]. The American Association of Poison Control Centers announces data for the USA for 2003 where out of a total of 145 394 caustic poisonings there were 22,000 (15.13%) poisonings with acids, 50,500 (34.7%) poisonings with bases, 16,272 (11.19%) poisonings with peroxides, 54,300 (37.3%) with chlorine hydrogen acid, and 2,322 (1.59%) poisonings with phenols or phenol compounds [14, 17, 20, 21]. Essential data are that acute peroral corrosive poisonings constitute 8–10% of the total number of poisonings [15, 22], 18–80% of the complications [16, 17, 18, 19], and 10–38% of mortality [10, 12]. The data for our country are comparable and show that the corrosive poisonings constitute 15–18% of the total number of poisonings, 14–27% of the complications, and 10–15% of the mortality [29, 30].

The injuries in the mouth, throat, oesophagus, stomach and duodenum can be either reversible or irreversible. The damaged mucous membrane, the sub-mucus and muscle layer regenerate only with great difficulty because of the surrounding inflammation, necrosis and secondary complications. Ensuing fibrosis and the organization of the tissues as well as creation of growth or circular

stenosis greatly disturb the function of the GIT (impeded peristaltic and passage), [3, 4, 21].

The most frequently misused substances are acids (HCl, acetic acid, sulphuric acid), and bases (NaOH), while oxidants, phenols and their compounds are rarely used. In our material, poisonings with chlorine hydrogen acid (HCL) dominate, in contrast to the USA and the European countries, where poisonings with bases (NaOH) and materials for sanitary maintenance are more frequent. In the literature they can be found under the general name of "lye" [16, 29].

The poisonings can be suicidal or accidental. They are more frequently suicidal, while more rarely accidental in previously alcoholic intoxicated patients [13]. In countries with a lower degree of health education the corrosive poisonings are more frequently suicidal [13, 15], while in countries which are more developed accidental poisonings are more frequent. They usually appear in a state of alcoholic intoxication and in patients having a lower level of education and lower social status, as is confirmed by our set of patients, too [7, 8].

In 10 to 30% of the patients with severe oesophageal post-corrosive burns there are no local changes in the mouth and pharynx. One large study showed the appearance of 37% of oesophageal injuries of second and third degree in patients who did not have obvious orrho-pharyngeal injuries [19, 20, 27]. On the other hand, it was stated that in 70% of the patients who had severe orrho-pharyngeal injuries there were no significant oesophageal burns. Therefore, injuries to the mouth and pharynx are not certain indicators for possible oesophagal injuries [22, 28, 31]. The major general manifestations in this type of lesion of inflammatory character are connected to the acute phase by the appearance of hypovolemia, dehydration and shock condition, adynamia and exaltation, water-electrolyte and alkaline-acid misbalance, increased body temperature, secondary infections in the damaged tissues, etc. [16, 17]. However, there are opposing statements in studies made regarding severe late complications in patients where there was a lighter degree of damage and an accidentally ingested corrosive substance [28].

Late complications are the major problem concerning acute corrosive poisonings, as well as a frequent cause of patients' permanent invalidity. In the last few years, sophisticated diagnostic methods and treatment have drastically decreased the percentage of late complications connected to acute corrosive poisonings and injuries of the upper part of gastrointestinal tract [21, 30, 32]. The most frequent late complications are oesophageal strictures and stenosis, stenosis of the antrum and pyloric part of the stomach, oesophagus and stomach cancer [3, 20]. Strictures and stenoses can appear three weeks after the corrosive ingestion, during the first three months, or even after a year according to some authors. The ingestion of liquid corrosives indicates stenoses more frequently than corrosives as a crystal substance [19]. Antrum and pylorus stenoses are

indicated by the appearance of symptoms such as, satiety, disgust, vomiting, and loss of body weight. They appear less frequently than oesophageal stenoses and most often at 5 to 6 weeks after the ingestion. According to some authors they can even appear after several years [15, 27]. The latent period of the corrosive substance ingestion and the appearance of cancer can be 50 to 60 years. According to some studies, 3% of oesophageal cancers give data of previous caustic substance ingestion. The most frequent localization is at the level of the carina, and the prognosis of these oesophageal cancers is better than that of oesophageal cancers with other etiologies [1, 31]. The appearance of stomach cancer is a very rare complication after corrosive ingestion.

The high percentage of late complications is noticeable in the data presented in our study (19.3%). In our series, oesophageal post-corrosive stenosis was found in 31 patients (6.0%), antrum and pylorus post-corrosive stenosis in 42 patients (8.1%) and combined post-corrosive stenosis of oesophagus, antrum and pylorus in 27 patients (5.2%). The overall percentage (19.33%) is comparable with the other reports, ranging from 12% to 85% [2, 4, 7, 8, 10, 12, 23].

Results published in various studies show that stenoses after the first endoscopic and X-ray examination were found in 27.8% of the patients, i.e. in 25.8% of the patients intoxicated by acids and 66.6 % of the patients intoxicated by bases [3, 4]. The most frequent topographic localization of the stenoses was the distal part of the oesophagus and the antrum-pyloric region of the stomach where the percentage in both cases was 6.4%. In 16.7 % of the patients intoxicated by alkali, stenoses developed in the middle and distal part of the oesophagus. In another study, in acute patients intoxicated with alkali a high percentage (62.3%) of oesophagus and stomach post-corrosive sequels was found [9].

Despite the intensive therapeutic approach in a group of 430 patients, who were suicidal and/or accidentally intoxicated by corrosives and were followed over the course of several years, a high percentage of complications developed (40%). In another group of patients late post-corrosive complications developed in 32.7% (19 out of 58 patients) [12]. Ingestion of alkali most frequently causes oesophageal stenosis (up to 85%), while in acid intoxications oesophageal stenosis is rare, i.e. from 10 to 15% [30, 32]. The most frequent complication after acute poisoning by acids was pyloreal stenosis (up to 25%), [7, 8].

The mortality in our study was 8.7% (45 patients). This is comparable with the reported mortality of around 10% [7, 8], and concerning the particular studies dealing with corrosive poisonings, it varies from 4% to 12% [1, 3, 26, 29, 30). The most frequent reasons for early death in acute poisonings with corrosives are tracheal necroses, mediastinitis and massive metabolic acidosis [16, 17].

Prognosis

Prognosis in acute corrosive poisonings is variable and depends on the degree of oesophageal and stomach damage, as well as the previous condition of the general health of the patient. In addition, acute corrosive poisonings are known to have the highest mortality because of the consequences of perforation and the development of mediastinitis.

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Р е з и м е

ЕПИДЕМИОЛОГИЈА НА ТЕШКИТЕ ТРУЕЊА ПРЕДИЗВИКАНИ СО ИНГЕСТИЈА НА КАУСТИЧНИ СУПСТАНЦИ

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Akutnite truewa so korozivni supstanci mo`at da predizvikaat te{ki hemiski povredi na gorniot del na gastrointestinalniot trakt, naj~esto lokalizirani na ezofagusot i `eludnikot. Vo slu~aj na pre`ivuvawe vo akutnata faza na trueweto, regenerativniot odgovor mo`e da rezultira so ezofagealna i/ili gastri~na stenoza i zgolemen rizik od karcinom na ezofagusot i `eludnikot.

Vo ovaa studija e obraboten sedumgodi{en klini~ki materijal (2000‡2006) so vkupno 517 pacienti lekuvani na Klinikata za urgentna interna medicina i toksikologija pri Univerzitetskiot klini~ki centar vo Skopje, Republika Makedonija.

Evaluacijata na kausti~nite truewa e sledena vrz osnova na anamneza i heteroanamneza, fizikalen pregled na pacientot i evidentirawe na lokalnite promeni na usnata sluzoko`a, jazikot, nepcata, grloto.

Koristeni se slednite tehniki na vizualizacija: urgentna ezofagogastroduodenoskopija vo prvite 24 ~asa po ingestijata, kontrolna ezofagogastroduodenoskopija (15 odnosno 25 dena po ingestijata), rentgenografija na ezofagus, gaster i duodenum so gastrografin (25‡30 dena po ingestijata), kako i rutinski laboratoriski isleduvawa (krvna slika, urea, kreatinin, enzimski, proteinski i lipiden status, serumski transferin, itn.), sledewe na aktuelnata telesna te`ina, t.e. body mass index (БМИ).

Rezultatite poka`aa deka dominiraat pacienti od `enski pol [n = 368 (71,79%), p < 0,001], hristijanska veroispovest [n = 263 (50,87%), p > 0,05)], so sredno obrazovanie [n = 322 (62,28%), p = 0,001] и хлороводородна киселина како најчесто злоупотребувано средство

[n = 245, (47,38%) p > 0,05]. Најголемиот број на пациенти при првиот ургентен езофагогастро-дуоденоскопски преглед имаат II A (n = 190) и II B (n = 136) степен на оштетување (X2 = 44,0, p < 0,05), а со стенози на езофагус и желудник се испишани 100 пациенти (19,34%). Високиот процент на стенози е мајорен фактор на ризик за појава на инвалидитет кај овие пациенти. Констатиран е и висок морталитет од 8,69% (p > 0.05).

Од 517 пациенти 62 (11,99%) биле труења со концентрирана оцетна киселина. Кај 37 пациенти (59,67%) како акутна компликација се развила акутна бубрежна инсуфициенција. Заради компликации од нарушување на бубрежната функција починале 4 (6,5%) пациенти, а кај 93,5% исходот бил воспоставување на бубрежната функција. Во текот на третманот пациентите биле третирани покрај другата терапија и со хемодијализа (ХД). Направени се вкупно 138 хемодијализи (3.7% по пациент).

Акутните корозивни труења претставуваат сериозен социо-економски и дијагностичко-терапевтски проблем. Најчесто се јавуваат кај работоспособната популација, во најкреативниот и најефективен животен период. Скапото лекување економски ја оптоварува општествената заедница и може да предизвика тежок инвалидитет кај пациентите. И покрај современото лекување и напорите за намалување на морталитетот и доцниот морбидитет, интоксикациите со корозивни средства остануваат тежок медицински проблем.

Клучни зборови: каустични труења, езофагогастродуоденоскопија, езофагеални и желудочни корозивни повреди.

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