ORIGINAL PAPER

Post-corrosive Late Complications in Esophagus and Stomach – Role of the Esophageal Rest

Andon Chibisev
University Clinic for Toxicology, Clinical Centre, Skopje, Republic of Macedonia

Acute corrosive poisonings cause severe chemical injuries of the upper gastrointestinal tract, the most common location being the esophagus and the stomach. There are different opinions concerning the question of taking food and liquids by mouth immediately after caustic ingestion. This prospective study comprised 146 patients aged between 14 and 75 years divided in two groups. In the examined group prevailed those with esophagitis gr.IIb (n=36; 54,54 %), esophagitis gr.III (n=30; 45,45 %), gastritis gr. IIb (n=42, 63,63 %) and gastritis III (n=24;36,36%). In the controlled group prevailed those with esophagitis gr III (n= 52; 65 %) and esophagitis gr IIb (n= 28; 35 %), gastritis gr. IIb(n= 55; 68,75 %)and gastritis gr III (n= 25; 31,25 %). Analysis of the results has shown a high percentage of esophageal stenosis in both groups 25 days after poisoning (31.82% v.s 43.75%), three and six months after poisoning (36.36% v.s. 52.50%) and also gastric injuries 25 days after the poisoning (37,88 % v.s. 46,25 %), three and six months after the poisoning (40,91% v.s 53,75%) In spite of the not significant difference, the results of our investigation have shown that the group with "esophageal rest" (NPO) had a smaller percentage of post-corrosive complications than the patients who were given food or liquids immediately after poisoning. **Key words:** caustic poisonings, caustic injuries, esophageal rest, post-corrosive stenosis

Corresponding author: Andon Chibisev, MD, PhD. University Clinic for Toxicology, Clinical Centre, Vodnjanska 17, 1000 Skopje, Republic of Macedonia, Tel: +389 2 3211 072, +389 2 3237 504, +389 2 3147 635, +389 70 387 040, +389 75 223 223, E-mail: toksikourgentna@gmail.com

1. INTRODUCTION

Acute corrosive poisonings appear as a result of ingestion of acids, bases, oxidants, heavy metal salts and other chemical substances. They cause chemical injuries of the upper gastrointestinal track, the most common location being the esophagus and the stomach. The patients present with a serious clinical picture; clinical examinations are difficult to be conducted and therapy and final outcome are often uncertain (1,2). The most serious lesions occur in the esophagus and the stomach since the poison remains there a long time due to the specific anatomic structure of these organs. 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 and gastric cancer (3,4).

In establishing the diagnosis and therapeutic approach of corrosive poisonings, the severity of the post-corrosive endoscopic changes of the esophagus, stomach and duodenum is of major importance. It is detected with esophagogastroduodenoscopy, which is conducted 12-24 hours after ingestion of the corrosive agent (5,6).

We usually use the classification of endoscopic post-corrosive injuries in the upper GIT suggested by Kikendall:

- First grade: erythema and edema of the mucosa;
- Second (A) grade: erosions, blisters, superficial
- ulcers (transversal and linear), exudation, hemorrhage;
- Second (B) grade: circumferential lesions;
- Third grade: multiple deep brownish-black or grey ulcerations and necrosis;
- Fourth grade: perforation (7).

Severity of the lesions depends on the nature, quantity and concentration of the corrosive substance, and the duration of exposure . The damaged mucosa, submucosa and muscle layer regenerate with great difficulty because of the surrounding inflammation, necrosis and secondary complications. Tissue fibrosis, adhesions or circular stenosis appear, which greatly disturb the normal functioning (impeded peristaltic, impeded passage). All these complicate the entire general condition of the patient, including inadequate normal food intake, loss of body weight, prostration, cachexia (8).

There are different opinions concerning the question of taking food and liquids by mouth immediately after caustic ingestion. Many authors recommend NPO (nill per os) or the so-called

"esophageal rest" until the first endoscopic control (10 – 15 days). During the "rest", the patient is fed completely parenterally by peripheral or central vein, or enterally by nasogastric or nasoenteral tube, gastrostoma or jejunostoma (9).

There is another group of authors that recommend taking liquids 48 hours after ingestion if the patient can swallow his/her saliva (10).

The aim of this paper was to present the influence of the esophageal rest i.e. NPO in development of post-corrosive stenosis in the upper gastrointestinal tract.

2. MATERIAL AND METHODS

This prospective study included a group of 146 patients, at the age between 14 and 75 years, hospitalized and treated at the University Clinic of Toxicology in Skopje in the period 2007-2009. The patients were divided in two groups:

Group 1: consisted of 66 patients, with mean age in the interval 42.9 ± 16.5 years, who were fed enterally (nasojejunal tube or feeding enterostoma) and were not given food by mouth in the first 10-15 days (NPO or esophageal rest). This was the examined group.

Group 2: consisted of 80 patients,with mean age in the interval 32.9 ± 15.6 years, who were given liquid food by mouth in addition to parenteral nutritional support in the first 48 hours after admission to the hospital. This was the control group.

Follow-up period lasted for 6 months.

In all patients diagnostic esophagogastroduodenoscopy was done in the first 12-14 hours after caustic ingestion, control esophagogastroduodenoscopy 15-25 days, three or six months after caustic ingestion. During urgent esophagogastroduodenoscopy in patients of group 1 with II B grade of injury nasojejunal tube was inserted under endoscopic control for post-pyloric nutrition. In patients with III grade of injury, after consulting an abdominal surgeon, a feeding enterostoma was implanted laparoscopically in the first 96 hours for artificial post-pyloric nutrition. Patients were analyzed according to their clinical findings, gender, way of poisoning (suicidal and accidental), chemical structure of the corrosive agent (acids, alkalis, other caustic substances).

Category	Admission		15 days after ingestion		25-30 days		3 months		6 months	
	E.R. (n)(%)	L.D. (n)(%)	E.R. (n)(%)	L.D. (n)(%)	E.R. (n)(%)	L.D. (n)(%)	E.R. (n)(%)	L.D. (n)(%)	E.R. (n)(%)	L.D. (n)(%)
Esophagitis	/	/	51 (77,27)	69 (87,25)	3 (4,55)	4 (5,0)	/	/	/	/
Esophagitis gr.II b	30 (45.45)	42 (52,50)	5 (7,58)	7 (8,75)	1 (1,52)	9 (11,25)	/	/	/	/
Esophagitis gr.III	36 (54.55)	38 (47,50)	8 (12,12)	4 (5,0)	1 (1,52)	2 (2,5)	/	/	/	/
Stenosis	/	/	1 (1,52)	/	21 (31,82)	35 (43,75)	24 (36,36)	41 (51,25)	24 (36,36)	42 (52,50)
Normal finding	/	/	1 (1,52)	/	40 (60,61)	30 (37,50)	42 (63,64)	39 (48,75)	42 (63,64)	38 (47,50)
Total	66(100)	80(100)	66(100)	80(100)	66(100)	80(100)	66(100)	80(100)	66(100)	80(100)

Table 1. Endoscopy/esophagus (esophageal rest v.s. liquid diet) E.R.- esophageal rest L.D.- liquid diet

Category	Admission		15 days after Ingestion		25-30 days		3 months		6 months	
	E.R. (n)(%)	L.D. (n)(%)	E.R. (n)(%)	L.D. (n)(%)	E.R. (n)(%)	L.D. (n)(%)	E.R. (n)(%)	L.D. (n)(%)	E.R. (n)(%)	L.D. (n)(%)
Gastritis	/	/	51 (77,27)	60 (75,0)	4 (6,06)	3 (3,75)	1 (1,52)	/	1 (1,52)	/
Gastritis gr.II b	42 (63,64)	46 (57,50)	10 (15,15)	11 (13,75)	1 (1,52)	2 (2,5)	/	/	/	/
Gastritis gr.III	24 (36,36)	38 (47,50)	3 (4,55)	9 (11,25)	/	/	/	/	/	/
Stenosis antri	/	/	/	/	4 (6,06)	9 (11,25)	5 (7,58)	8 (10,0)	4 (6,06)	10 (12,5)
Stenosis antropylori	/	/	1 (1,52)	/	10 (15,15)	13 (16,25)	11 (16,67)	16 (20)	12 (18,18)	16 (20)
Stenosis pylori	/	/	/	/	11 (16,67)	15 (18,75)	11 (16,67)	17 (21,25)	11 (16,67)	17 (16,67)
Normal finding	/	/	1 (1,52)	/	36 (54,54)	38 (47,50)	38 (57,58)	39 (48,75)	38 (57,58)	37 (46,25)
Total	66(100)	80(100)	66(100)	80(100)	66(100)	80(100)	66(100)	80(100)	66(100)	80(100)

Table 2. Endoscopy/gaster (esophageal rest v.s.liquid diet) E.R.- esophageal rest L.D.- liquid diet

The following statistical methods have been applied:

- Chi-square test (X^2 test) was used to determine the significance of difference in the onset of stenosis in the examined and control groups (post-corrosive esophagitis gr. IIb and III and post-corrosive gastritis gr. IIb and III);
- Relative risk (RR) was used to determine the risk for onset of stenosis in the examined and control groups (post-corrosive esophagitis gr. IIb and III and post-corrosive gastritis gr. IIb and III).

3. RESULTS

The examined group consisted of 66 patients, of whom 15 (22.72%) were males and 51 (77.27%) females; the youngest patients being 18 years old and the oldest 71 years old (± 95.000 Confid. Interval from 37.3 to 48.4 years.).

Days when patients received no food by mouth (NPO) varied in the interval 10.39±1.49 days, minimum 8 days and maximum 14 days.

The control group consisted of 80 patients, of whom 16 (20%) were males and 64 (80%) females. The youngest patients were 14 years old and the oldest 75 years old (± 95.000 Confid. interval from 29.4 to 36.3 years).

These patients were given liquid food by mouth 48 hours after esophageal rest.

The largest number of the examined group of patients – 43 (65.15%) ingested HCl, 15 (22.72%) used NaOH and 8 (12.12%) CH_3COOH.

The majority of patients – 61 (92.42%) had suicidal intent and 5 (7.57%) ingested caustic agent accidentally.

In the control group of patients, prevailed those who ingested hydrochloric acid – 51 (63.75%), 13 (16.25%) used acetic acid, 9 (11.25%) ingested sodium hydroxide whereas 7 (8.75%) sulphuric acid.

Analysis of the results in both groups of patients showed a high percentage of esophageal stenosis 25 days

Variable	Stenosis	Normal	RR	95%CI
15 days				
EG	1	65		
CG	0	80	Infinity	Nan<O.R.<Infinity
25-30 days				
EG	21	45		
CG	35	45	0.73	0.47-1.12
3 months				
EG	24	42		
CG	41	39	0.71	0.49-1.04
6 months				
EG	24	42		
CG	42	38	0.69	0.47-1.01

EG- examined group; CG- control group; RR-Risk Ratio; CI- Confidence interval

Tabela 1a. Determination of risk for occurence of stenosis in patients with post-corrosive injuries (esophagitis IIA and III) in the examined and control group

Variable	Stenosis	Normal	RR	95%CI
15 days				
EG	1	65		
CG	0	80	Infinity	Nan<O.R.<Infinity
25-30 days				
EG	24	36		
CG	37	43	0.86	0.59-1.28
3 months				
EG	27	39		
CG	41	39	0.80	0.56-1.14
6 months				
EG	27	39		
CG	43	37	0.76	0.53-1.08

EG- examined group; CG- control group; RR-Risk Ratio; CI- Confidence interval

Tabela 2a. Determination of risk for occurence of stenosis in patients with post-corrosive injuries (gastritis IIA and III) in the examined and control group

after poisoning (31.82% vs 43.75%) and three and six months after poisoning (36.36% vs 52.50%) (Table 1).

Analysis of the results in both groups of patients has shown a large percentage of post-corrosive gastric injuries 25 days after caustic ingestion (37.88% vs 46.25%) and three and six months after caustic ingestion (40.91% vs 53.75%) (Table 2).

Table 1a. shows the results of the risk for onset of post-corrosive injuries (esophagitis IIB and III) in the examined and control groups of patients.

Fifteen days after initiation of therapy, for chi-square=1.22 and p >0.05 (p=0.27), there was no significant difference in the distribution of the results between the examined and control group.

After administration of therapy for 25-30 days, the chance for occurence of stenosis in patients fed by tubes was 0.73 times smaller / RR=0,73 (95%CI 0.47-1.12)/, in comparison with patients given liquid diet, although the difference was not significant.

Three months after treatment, the chance for occurence of stenosis in patients feb by tubes was 0.71 times smaller / RR=0,71 (95%CI 0.49-1.04)/, in comparison with patients given liquid diet, although the difference was not significant.

After 6 months, the chance for occurence of stenosis in patients feb by tubes was 0.69 times smaller / RR=0,69 (95%CI 0.47-1.01), in comparison with patients given liquid diet, although the difference was not significant.

Table 2a. presents the results of the risk for onset of stenosis in patients with post-corrosive injuries of the stomach (gastritis IIB and III) in the examined and control groups of patients.

Fifteen days after initiation of the therapy, for chi-square=1.22 and p >0.05 (p=0.27), there was no significant difference in the distribution of the results between the examined and control group.

After administration of therapy for 25-30 days, the chance for occurence of stenosis in patients fed by tubes was 0.86 times smaller / RR=0,86 (95%CI 0.59-1.28)/, in comparison with patients given liquid diet, although the difference was not significant.

Three months after treatment, the chance for occurence of stenosis in patients feb by tubes was 0.80 times smaller / RR=0,80 (95%CI 0.56-1.14)/, in comparison with patients given liquid diet, although the difference was not significant.

After 6 months, the chance for occurence of stenosis in patients feb by tubes was 0.76 times smaller / RR=0,76 (95%CI 0.53-1.08), in comparison with patients given liquid diet, although the difference was not significant.

4. DISCUSSION

Corrosive agents cause tissue destruction due to coagulation or liquefaction necrosis, which intensity depends on the type and concentration of the corrosive agent, duration of exposure, quantity of the ingested agent, etc. (11). A retrospective study conducted in 239 patients who ingested sodium hydroxide (NaOH) detected post-corrosive esophagitis in 89.3% of the patients and esophageal or gastric stenosis in 72.6%. One percentage of the patients died in the acute phase of poisoning (in the first 24 h) and 1.4% in the chronic phase of the poisoning. It is interesting to emphasize that the incidence of stenosis in female patients was 80.8% and in male patients 62.5%. Percentage of stenosis in patients who ingested a larger amount of corrosive agent (two or several spoonfuls) was 93.6%; post-corrosive carcinoma of the upper GIT was found in 1.8% of the patients, fistulas in 9.9% and perforation in 4.6% of the patients (12).

Intensity and mucous damage of the upper GIT in caustic poisonings are of huge importance in decision making on the therapeutic approach. The majority of authors think that hyperalimentation (enteral or parenteral) and antibiotic therapy have the biggest effect in successful healing. In contrast to post-corrosive injuries of grade I and II A that usually do not develop post-corrosive complications, 50-70% of patients with II B grade of injury develop stenosis in the first 6 months after ingestion and all (100%) of patients with III grade of injury (13).

Extensive damage of the upper gastrointestinal tract of grades II A and III hinder physiological nutrition in these patients and, within a short period of time, they fell into a severe general condition due to hypercatabolic state and negative alkali balance. Artificial nutrition is life-maintaining therapy in patients who cannot take food and are disposed to a risk of malnutrition. The type of the artificial nutrition depends on the degree of esophageal or gastric damage (14).

In many studies, patients with II B and III grades of injury are recommended the so-called esophageal rest. It may be possible only if the patient does not take food by mouth (NPO or nill per os). During the "rest", the patient is fed enterally by nasogastric or nasoen-

teral tube, gastrostoma or jejunostoma and parenterally by peripheral or central vein. The positive effect of not taking food by mouth for a certain period of time is explained by the fact that food particles do not enter granulocytes of the esophageal wall and do not exacerbate the inflammation which is in a positive correlation with late post-corrosive complications (15,16).

One study which examined 522 patients demonstrated positive effects of the esophageal rest in the first 10 days after caustic ingestion regarding the percentage of the severe late post-corrosive complications (17). Another study performed on 118 patients examined the relation of the esophageal rest with the percentage of late post-corrosive complications and revealed a positive relation between not taking food by mouth (NPO) and number of patients who had stenosis of the upper GIT as a final outcome of the poisoning (18).

Many authors recommend avoiding food intake by mouth (NPO) until the 10th day after ingestion and some other authors recommend it until the 15th day, that is, until the first endoscopic control (19). There is a group of authors who advocate taking liquids 48 hours after corrosive ingestion if the patient can swallow his/her saliva. These authors also think that 48 hours after corrosive ingestion the patients can take food by mouth (20). Deskin R. (21) supports the so-called esophageal rest in patients with acute corrosive poisonings. This rest is possible if the patient does not take food by mouth (NPO or nill per os). During the rest, the patient is fed by nasoenteral tube, gastrostoma or jejunostoma. This author explains that taking food by mouth exacerbates the infection of GIT post-corrosive burns and thus, delays and complicates the healing of the lesions. If enteral nutrition is impossible, then he advises TPI.

Kikendal (22) advises the so-called esophageal rest of minimum 10 days since the endoscopic diagnosis of the poisoning and permanent control of the nutritional status of the patient for maintenance of the good general condition. Eric Kardon (23) does not recommend taking food by mouth (NPO) in acute corrosive poisonings until the first endoscopic control (between 15 and 20 days). Katzka A. David (24) thinks that in all corrosive poisonings of II B and III grades of injury of upper GIT, it is necessary to implant feeding tube (jejunostoma) for nutritional support and to avoid food intake by mouth (NPO) for 15 days.

One study examined 118 children with severe post-corrosive esophageal and gastric burns who, in addition to the usual therapy, received nothing by mouth for 7 days in order to prevent colonization of bacteria on the necrotic parts and to reduce the possibility of infection. Seventeen percentages of them developed post-corrosive stenosis. Since physiological nutrition was not possible, feeding gastrojejunostoma was installed until the adequate surgical treatment (25).

5. CONCLUSION

Acute corrosive poisonings are a serious clinical, socio-economic and diagnostic-therapeutic problem, which, in spite of the modern sophisticated diagnostics and treatment, cause a serious invalidity in patients. These poisonings are most common in subjects who are in their most creative period of life; they burden the social community because of the expensive diagnostic and therapeutic programs and prolonged hospital stay.

In spite of the not significant difference of the results obtained in our study, it was shown that the group of patients with "esophageal rest" (NPO) had a smaller percentage of post-corrosive complications than the patients who were given food or liquids immediately after caustic ingestion. This might be a result of avoiding additional injuries during NPO status as well as of less frequent infections that would compromise the healing of post-corrosive injuries.

REFERENCES

1. Allakhverdian AS, Maurin VS, Antisecretory therapy for prevention of stenosis of bouginage after burn of esophageal strictures. Eksp Klin Gastroenterol, 2003;114(4):36-9.
2. Andreoni B, Biffi R, Padalino P, Marini A, Marzona L, Belloli S, Farina ML, Tiberio G. Artificial nutrition in the management of lesions caused by caustic ingestion. Chir Ital, 1994;46(6):42-8.
3. Zwischenberger, Joseph B. Clare Savage, Bidan A, Surgical Aspects of Esophageal Disease . Am J Respir Crit Care Med, April 2002;165(8):1037-40.
4. Arévalo-Silva C, Eliashar R, Wohlgelernter J, Elidan J, Gross M. Ingestion of caustic substances: a 15-year experience Laryngosc, 2006;116(8):1422-6.
5. Berthet B, Bernardini D, Lonjon T. Treatment of caustic stenoses of the upper digestive tract. Chir (Paris), 1995;132 (11): 447-50.
6. Abakumov MM, Kostiuchenko LN, Kudrichoba NE. Enteral infusion-nutrional correction of homeostasis in patients with postburn cicatrial stenosis of the esophagusand stomach.Vestn Khir Im II Grek, 1999;155(5):30.
7. Alinejad A. Caustic injury to upper gastrointestinal tract,Shiraz university of medical sciences, Depertment of internal medicine, Available from:pearl.sums.ac.ir/semj/vol4/jan2003/causticinj.htm
8. Atabek C, Surer I, Demirbag S, Caliskan B, Ozturk H, Cetinkursun S. Increasing tendency in caustic esophageal burns and long-term polytetrafluorethylene stenting in severe cases: 10 years experience. J Pediatr Surg, 2007 Apr;42(4):636-40.
9. Post-corrosive injuries of upper gastrointestinal tract, Chibishev A, Simonovska N, Shikole A, Contributions, 2010 Jul;31(1):297-316.
10. Ramasamy K, Gumaste VV. Corrosive ingestion in adults, J Clin Gastroenterol, 2003 Aug,37(2):119-24.
11. Chibishev A, Chibisheva B, Bozinovska C, Naumovski J. Oesophageal and gastric stenoses are common complications after acute oral poisoning with corrosive agents. Maced J Med, 2005;51(1-2):139-46.
12. Mamede RC, De Mello Ficho FV. Treatment of caustic ingestion: an analysis of 239 cases. Dis Esophagus, 2002;15 (3):210-3.
13. Conforto F, Gercitano M, Tanga I. Emergency treatment of esophago-gastric lesion in caustic ingestion patiens. Critical Care, 2004,(Suppl 1)8:P 284.
14. Di Costanzo J, Noirclerc M, Jouglard J, Escoffier JM, Cano N, Martin J, Gauthier A. New therapeutic approach to corrosive burns of the upper gastrointestinal tract. Gut, 1980 May;21(5):370-5.
15. Kochhar R, Poornachandra KS, Puri P, Dutta U, Sinha SK, Sethy PK, Wig JD, Nagi B, Singh K. Comparative evaluation of nasoenteral feeding and jejunostomy feeding in acute corrosive injury: a retrospective analysis. Gastrointest Endosc, 2009 Nov;70(5):874-80.
16. Katzka A, David MD. A standardised protocol for the acute management of corrosive ingestion in children. J Pediatr Surg. 2005;40(7):1214-5.
17. Cibisev A, Nikolova-Todorova Z, Bozinovska C, Petrovski D, Spasovski G. Epidemiology of severe poisonings caused by ingestion of caustic substances. Prilozi, 2007;28(2):171-83.
18. Andreoni B, Farina ML, Biffi R, Crosta S. Esophageal perforation and caustic injury, emergency menagment of caustic ingestion. Dis Esophagus, 1997;10 (2):95-100.
19. Sarfati E, Gossot D, Assens P. Management of caustic ingestion in adults. British Journal of Surgery, 2005;74 (2):146–8.
20. Zabelegui A, Mijan de la Torre. Severe gastroesophageal lesions due to caustics; the role of nutritional support. Nutr Hosp, 1995;10 (6): 364-7.
21. Deskin R. Caustic ingestion. com [homepage on the Internet]. U TMB, Grand Rounds Dept. of Otolaryngology; 1995 [updated 2001 april 17; cited 2010 june 30].Available from:www.utmb.edu/otoref/grnd/ Aerodigestive-Tract-2001-04.htm
22. Kikendal JW. Caustic ingestion injuries. Gastroenterol Clin North Am, 1991; 20(4): 847-57.
23. Kardon E. Caustic ingestion, com [homepage on the Internet]. Emergency Medicine Toxicology. [updated 2010 May; cited june 2010]. Available from: emedicine.medscape.com
24. Katzka A, David MD. Caustic Injury to the Esophagus. Current Treatment Options in Gastroenterology, 2001;1(4):59-66.
25. Schmittenbecher P. A standardised protocol for the acute management of corrosive ingestion in children. J Pediatr Surg, 2005;40(7):1214-5.