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Full Length Research Paper

Acid and alkali burns of the esophagus: An experimental study

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The purpose of the study was to evaluate esophageal burn in a rat model using varying concentrations of acid and alkali substances commonly used in daily life. Wistar Albino rats were used in this experimental model (n = 35). One control group and four working groups were designated with 7 animals in each. The control group was given serum physiologic into the esophagus; Groups I and II were administered sodium (Na) hypochloride (acid) in concentrations of 2.5 and 5%, respectively; and Groups III and IV were administered Na hydroxide (alkali) in concentrations of 2.5 and 5%, respectively. After 24 h, samples were taken from the esophagus and the damage was evaluated in submucosa, muscularis mucosa and tunica muscularis, and the total burn scores were determined. Total burn score in the control group was 0.28±0.48; in Group I 2.28±0.95, in Group II 2.43±0.78, in Group III 3.00±0.57 and in Group IV 3.57±0.53. Significant differences were determined in total burn score between all study groups compared with the control group. There were significant differences between Groups I and IV and between Groups II and IV regarding muscularis damage ($p<0.05$). When the data were evaluated, there was more definite esophageal burn in both acid and alkali groups than in the control group. When the acid and alkali groups (of same concentrations) were compared, damage in the alkali group was greater. Also, the paper concluded that the type of substance given to esophagus as well as its concentration is important in esophageal burns.

Key words: Alkali, acid, corrosive esophageal burns, rat.

INTRODUCTION

Alkali substances are the most frequent cause of corrosive burns of the esophagus. In the pediatric group, 90% of burns are caused by alkali substances and 10% by acid substances (Janousek et al., 2006; Ozel et al., 2004). Household cleaning products such as laundry soda and different detergents generally contain alkali substances. While ingestion of alkali substances causes liquefaction necrosis in esophageal mucosa and submucosa, acidic substances cause coagulation necrosis (Tiryaki et al., 2005). Ingestion of corrosive substances in children is generally accidental; adults ingest such substances by mistake or in a suicide effort. Ingestion of strong corrosive substances, especially alkali substances can cause clinical symptoms ranging from narrowing of esophagus to acute perforation and even death (Otcu et al., 2003; Ekingen et al., 2005). Corrosive esophageal burns are an important problem both due to their frequency and serious clinical presentations. Our investigation addresses only esophageal burns in the acute period; long-term results were not evaluated. The aim of this study was to induce esophageal burn in rats using acid and alkali substances in commonly used concentrations and to evaluate the resultant burns. The

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Table 1. Scale of histopathologic evaluation.

Criteria	Score
Damage to the muscularis mucosa	
None	0
Present	1
Increase in submucosal collagen	
None	0
Mild (submucosal collagen at least twice the thickness of muscularis mucosa)	1
Marked (submucosal collagen more than twice the thickness of muscularis mucosa)	2
Damage and collagen deposition in tunica muscularis	
None	0
Mild (collagen deposition around the smooth muscle fibers)	1
Marked (same as mild with collagen deposition replacing some fibers)	2

data can shed some light in determining clinical approach and extent the period of the treatment in the patients who consume a corrosive substance.

MATERIALS AND METHODS

Wistar Albino rats, weighing 250 to 300 g and obtained from Cumhuriyet University Faculty of Medicine, Experimental Animals and Research Laboratory were used in this study. The study was approved by the local ethical committee at Cumhuriyet University. Two preliminary studies were conducted. The first was to determine the level of the esophagus at which the corrosive substance would be administered. For this purpose, the thorax and abdomen were dissected surgically and the distance between the mouth of the rat and cervical esophagus was measured. This length was marked on an 8-French feeding tube (infant feeding tube, Ramsons, India). In the study, corrosive substances were administered using the 8-French feeding tube inserted to this predetermined level. The second preliminary study involved determining the period of greatest pathological damage after intake of the corrosive substance and 5 rats were used for this purpose with esophagus sections of one rat each examined on the first, second, third, fourth and fifth days following administration of corrosive substance. The greatest pathological damage was determined on the first day. Hence, for the purposes of the current study, it was decided to take the esophagus sections one day following burn inducement. Liquids containing acid and alkali which are generally available in markets were examined. The concentrations were determined in the Pharmacology Department of our Faculty; thus, the chemicals were used in the experiment at the same percentages as sold in markets. One control group and four working groups were formed of 7 rats each, for a total of 35 rats. Pure acid (sodium-Na hypochloride) and pure alkali (Na hydroxide) substances were diluted and 2.5 and 5% concentrations were prepared.

Serum physiologic (0.2 ml) was given to the control group, 2.5 and 5% acid (Na hypochloride) to Groups I and II, and 2.5 and 5% alkali (Na hydroxide) to Groups III and IV (0.2 ml corrosive substance to all working groups). After chemical administration, the feeding tube was withdrawn. Rats were not given any food over 24 h and were operated the following day under anesthesia [Xylazine (Rompun), Bayer 3 mg/kg i.m. and ketamine HCl (Ketas), Pfizer 90 mg/kg i.m.]. Mediastinum and abdomens of rats were opened and esophagus and stomach were excised.

Histopathological evaluation

Sections were taken 3 cm proximal to the gastro-esophageal junction for pathological evaluation. Esophageal tissue samples were fixed in 10% buffered neutral formalin for 24 h. After routine tissue sample process, paraffin-embedded tissue blocks were prepared. 5 µm sections of tissue samples were stained with hematoxylin-eosin (H&E) and with Masson's trichrome (MT) to evaluate changes in ligament tissue. All specimens were evaluated by the same pathologist who was blinded to the study groups. Histopathological evaluation was performed according to the criteria as shown in Table 1 (Turkyilmaz et al., 2005).

Statistical method

Statistical analysis was performed using SPSS software. Chi-square test and Fisher's chi-square test were used to evaluate data, and the importance of difference between percentages test, Kruskall-Wallis test and Mann-Whitney U test for independent groups. Data presented in the tables were determined as arithmetical mean±standard deviation, median value, and number of subject and percentage error level was taken as 0.05.

RESULTS

When the groups were evaluated histopathologically, total burn scores were determined as 0.28±0.48 in the control group; 2.28±0.95, 2.43±0.78 in Groups I and II (acid, 2 and 5%, respectively); and 3.00±0.57, 3.57±0.53 in Groups III and IV (alkali, 2 and 5%, respectively) (Figure 1). When Groups I, II, III and IV were compared with the control group, the differences in total burn score were significant ($p<0.05$). A correlation was determined in study groups between concentration and total score ($r =$

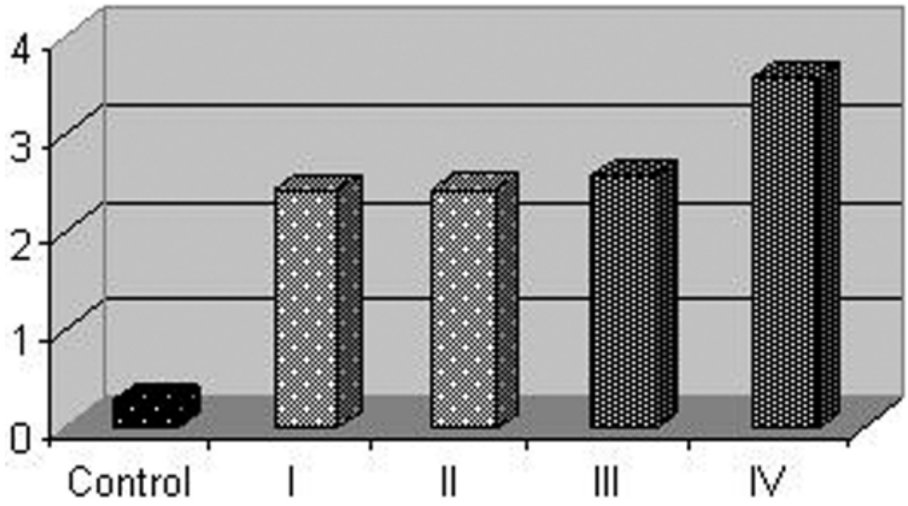

Figure 1. Total burn scores according to groups.

0.29; p = 0.139; p>0.05) such that total score increased in conjunction with concentration increase, but the increase was not important statistically. Muscularis mucosa damage according to groups was compared (Table 2). In the 'control group', two muscularis mucosal damage which might relate with feeding tube insertion was observed. While a significant difference was found between the control group and the other groups (p<0.05), there was no significant difference between the groups given alkali versus acid (p>0.05). The groups were evaluated according to submucosal damage (Table 2). The greatest damage was determined in Group IV and the least in Group I. Damage in esophagus slices is shown in Figures 2a and b. When the groups were compared regarding submucosal damage, differences between control group and Groups I and IV were significant (t = 2.37; p<0.05).

The difference between other groups was insignificant (t = 1.09; p>0.05). When the groups were evaluated regarding tunica muscularis damage, there was no damage in the control group and the greatest damage was determined in Group IV (Figure 3). Serious damage was not observed in any group. There were important differences in tunica muscularis damage between the control group, Groups III and IV (p<0.05). While the difference between Groups I and II was not significant (p>0.05), the difference between Groups I and IV and Groups II and IV was important (p<0.05).

DISCUSSION

When strong acid and alkali substances come in contact with live tissue, they produce chemical burn by causing dehydration in cells and coagulation of collagen and the other cell proteins (Turkyilmaz et al., 2005). The level of burn differs depending on type of tissue, period of contact, acid and alkali substance concentrations (Yukselen et al., 2005; Zhou et al., 2005). In our work, it was observed that the various acid and alkali substances available on the market can produce different burn levels. As shown in our study, all kinds of corrosive substances cause esophageal burn. Any event that may increase the damage after contact must be strictly avoided. In some studies, it was observed that alkali substances can pass to deep tissues, causing liquefaction necrosis and progressing to perforation (Cakmak et al., 1997). Thus, alkali substances may cause liquefaction necrosis and reach the muscle layer, and as the concentration increases, perforation may occur (Turkyilmaz et al., 2005). In our study, esophageal burn produced by alkali substances also reached the muscle layer. In Group IV, with the higher alkali concentration, more damage was observed when compared with the other groups. There was no perforation in any rat in this study. In our study, esophageal damage was evaluated in each layer of the esophagus. When groups were compared regarding level of muscularis mucosa damage, all groups had some damage compared with the control group, but damage was at the same level between groups. In fact, when the mucosa damage was evaluated, the least damage was determined in the 2.5% acid group and the greatest in the 5% alkali group (p<0.05). No serious damage in rat esophagus was determined except in the 5% alkali-administered group. Although corrosive esophageal burns are encountered often, treatment methods vary widely between clinics (Cheng et al., 2003). There is no common consensus regarding the time of esophagoscopy, steroid intake or the exact period of oral nourishment (Zhou et al., 2005; Avanoglu et al., 1998).

Table 2. Damage scores of the groups according to esophageal layers.

	MM		SM			TM		
Groups	**Damage (-) (%)**	**Damage (+) (%)**	**Damage (-) (%)**	**Damage (mild) (%)**	**Damage (marked) (%)**	**Damage (-) (%)**	**Damage (mild) (%)**	**Damage (marked) (%)**
Control	5 (71.4)	2 (28.6)	7 (100)	-	-	7 (100)	-	-
Group I	-	7 (100)	2 (28.6)	5 (71.4)	-	5 (71.4)	2 (28.6)	-
Group II	1 (14.3)	6 (85.7)	-	5 (71.4)	2 (28.6)	5 (71.4)	2 (28.6)	-
Group III	-	7 (100)	-	5 (71.4)	2 (28.6)	2 (28.6)	5 (71.4)	-
Group IV	-	7 (100)	-	3 (42.9)	4 (57.1)	-	7 (100)	-

SM: Submucosa, MM. Muscularis mucosa and TM: Tunica muscularis.

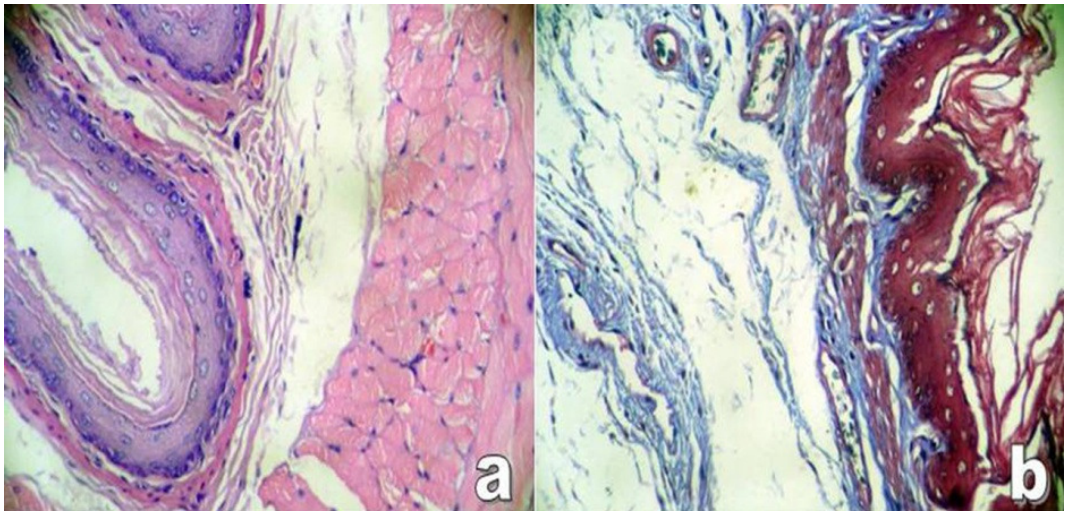

Figure 2. Normal rat esophagus. a: (H&Ex50), b: (MTx50).

Some authors are proponents of earlier esophagoscopy (de Jong et al., 2001), while other publications present the drawbacks of this approach. We do not prefer early-period esophagoscopy for various reasons.

In early esophagoscopy, the procedure is terminated at the level where the burn is first observed and there is no knowledge about distal damage. In the patient under anesthesia, the esophagus sphincter relaxes, and the corrosive substance can move back into the esophagus and increase the burn. Insertion of nasogastric tube also can be avoided for these reasons. The important point is determining whether or not perforation has occurred after intake of corrosive substance (Pelclova and Navratil, 2005; de Jong et al., 2001). If there is no perforation, the condition can be controlled. After this phase, the treatment can be planned to reduce the appearance of a later stricture. In our unpublished clinical series (n = 51), we have not encountered any perforation as well as in this experimental work. According to our limited experience, we can say that patient's history and physical examination

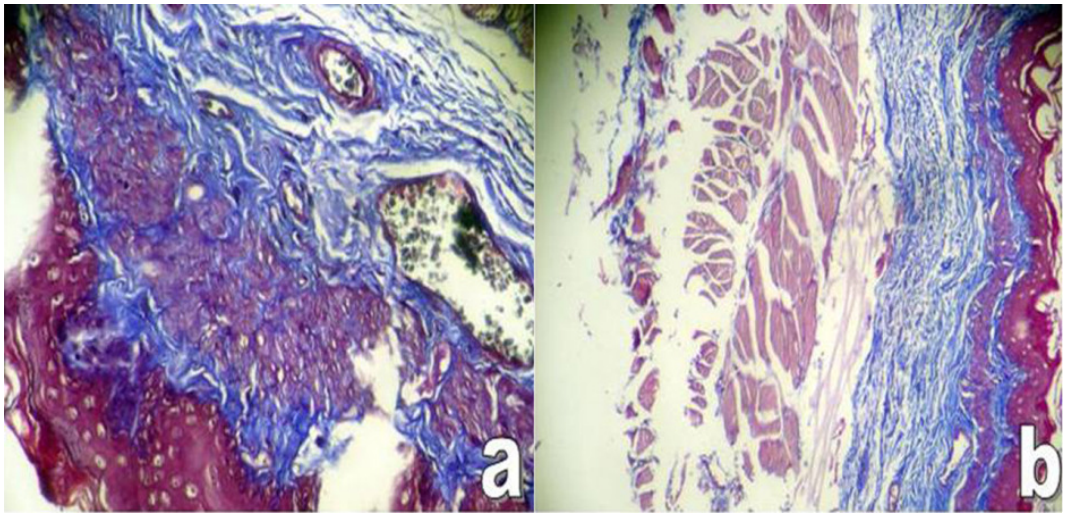

Figure 3. Increased collagen is seen from mucosa to tunica muscularis in Group IV. a: (MTx100) and b: (MTx50).

must be carefully checked. The invasive measurements should be avoided if possible. In Turkey, corrosive substances in liquid or crystal form are sometimes sold in water or beverage bottles. Sixty-nine patients with esophageal burns were observed and 74% of them had consumed the corrosive substance accidentally (n = 51). This practice of selling of corrosive substances can be improved by requiring the products to be sold in suitable and well-marked bottles.

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