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Management of gastrostomy to prevent perforation in acute severe corrosive esophagitis and gastritis: An experimental study

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Background/aims: *Symptomatic treatment is still the most commonly preferred treatment modality for acute severe esophagitis and gastritis. Clinical results of this treatment range from pathologies like stricture formation to loss of life. In our study, we aimed to demonstrate the effect of immediate gastrostomy in preventing perforation due to corrosive trauma.* ***Methods:*** *We used 32 rats in two study groups. In Group I (n: 16 rats), 1 ml of corrosive agent (10% NaOH solution) was administered and immediate gastrostomies were performed within 2 hours. In Group II (n: 16 rats), 1 ml corrosive agent (10% NaOH solution) was administered and the rats were treated symptomatically; no operation was performed.* ***Results:*** *Acute death was observed in 5 rats just after the corrosive agent was administered at the beginning of the study. Three rats from Group II died due to esophageal and gastric perforation within one week (25%). Necrosis was reported in 5 non-gastrostomized rats; however, no necrosis was observed in the gastrostomized group (p=0.037).* ***Conclusions:*** *Severe acute corrosive esophagitis and gastritis may be fatal. Furthermore, survivors may suffer from lifelong associated problems. From this study, we concluded that immediate gastrostomy in acute corrosive esophagitis and gastritis may play an important role in preventing necrosis and perforation risk.*

Key words: Corrosive agent, esophagitis, gastritis, perforation

Akut ciddi derecedeki koroziv özofajitis ve gastritiste perforasyonu önlemek için gastrostomi tedavisi: Deneysel çalışma

Amaç: *Akut ciddi derecedeki koroziv özofajitis ve gastritiste semptomatik tedavi hala en çok tercih edilen tedavidir. Bu tedavinin sonuçları darlık oluşumundan yaşamın kaybedilmesine kadar değişmektedir. Bu çalışmada koroziv travma sonucu perforasyonun önlenmesi için acil uygulanan gastrostominin etkisini göstermeyi amaçladık.* ***Yöntem:*** *İki grupta 32 rat kullanıldı. Grup I'de 16 rata 1 ml koroziv ajan (%10 NaOH solüsyonu) verildi ve 2 saat içerisinde gastrostomi gerçekleştirildi. Grup II'deki 16 rata ise 1 ml koroziv ajan (%10 NaOH solüsyonu) verildi ve ratlar semptomatik olarak tedavi edildi, operasyon uygulanmadı.* ***Bulgular:*** *Çalışmanın başlangıcında koroziv ajan verildikten hemen sonra 5 ratta akut ölüm gerçekleşti. Grup II'de bir hafta içerisinde özofageal ve gastrik perforasyondan dolayı 3 rat öldü. Gastrostomi uygulanmayan 5 ratta nekroz gözlendi. Gastrostomi uygulanan grupta nekroz gözlenmedi (p=0.037).* ***Sonuç:*** *Ciddi derecedeki akut koroziv özofajitis ve gastritis ölümcül sonuçlara neden olabilir. Daha sonraki zamanlarda yaşayabilenler eşlik eden problemlerden sıkıntı çekebilirler. Bu çalışmada akut koroziv özofajitis ve gastritiste nekroz ve perforasyon riskini önlemede acil uygulanan gastrostominin önemli bir rol oynayabileceğini bildiriyoruz.*

Anahtar kelimeler: Koroziv ajan, özofajitis, gastritis, perforasyon

INTRODUCTION

Corrosive esophagitis is inflammation and damage to the esophagus after ingestion of a caustic chemical. Similar to a burn, this injury may be temporary or may lead to permanent stricture

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(narrowing or stenosis) of the esophagus that is correctable only through surgery. Severe injury can quickly lead to esophageal perforation, mediastinitis and death from infection, shock and massive hemorrhage (due to aortic perforation).

Ingestion of a corrosive agent is still a life-threatening condition, and the actual incidence of this condition ranges from 1/5000 to 1/26000 in the United States. Damage to the esophagus and stomach by caustic agents depends on the type, concentration and form of those caustic agents and the duration of exposure (1,2).

The perforation rate in esophageal dilatation is reported to be 0.1–0.4% (3,4), but the risk of perforation increases in irregular, longer and high-grade strictures (5), with a reported perforation rate of up to 32% of patients after dilatation of caustic strictures (6,7).

According to the accepted general management scheme, patients who ingest corrosive agents must be hospitalized and treated symptomatically. Further management is determined according to the complications. Caustic-agent ingestion often results in pylorospasm and contractions in the stomach, causing retrograde ejection of the caustic agent back to the esophagus. On the other hand, the secondary cricopharyngeal muscle spasm causes propulsion of the caustic agent back to the stomach. This "seesaw" motion of the caustic agent aggravates both the esophageal and gastric burn (6,8). In severe cases, mediastinitis and peritonitis may be observed due to perforation and necrosis in the early stage. In these cases, a high mortality rate is reported. In patients without perforation, severe stenosis may develop, which causes distress over a period of time (in years) (1,2).

In this study, we aimed to demonstrate that perforation risk in acute severe corrosive agent ingestions may be prevented in rats with immediate gastrostomy, which can decrease the exposure time of the corrosive agent, if nasogastric tube and/or esophagoscopy cannot be performed.

MATERIALS AND METHODS

A total of 32 rats, weighing between 250 and 300 g, were used in this study. All the animals were handled according to the guidelines that complied with the Principles of Laboratory Animal Care of the National Society for Medical Research and the Guide for the Care and Use of Laboratory Animals formulated by the National Academy of Sciences (NIH publication 85-23, revised 1985). The Kirikkale University Ethical Committee on Animal Research approved this study.

Sodium hydroxide was the preferred corrosive agent because of its well-known effects as well as being the most commonly used corrosive agent in experimental studies (9, 10).

Operative Procedure

The rats were divided into two groups of 16 rats each. All the animals were premedicated with 10 mg/kg of intramuscular ketamine hydrochloride (Ketalar, Pfizer), and anesthesia was induced with 3 mg/kg of intramuscular xylazine.

The cap of an intravenous cannula was used as the esophageal tube. The corrosive agent was administered to the upper esophagus by means of this tube. Five rats died after the corrosive agent was administered in this period.

In Group I (n: 16), 1 ml of the corrosive agent (10% NaOH solution) was administered, and immediate gastrostomy was performed within 2 hours (h). In this group, laparotomy was performed followed by gastrostomy, and the gastric contents were consequently aspirated. The stomach and the esophagus were irrigated only once with isotonic solutions via gastrostomy. Subsequently, all the gastrostomies were closed.

In Group II (n: 16), 1 ml of the corrosive agent (10% NaOH solution) was administered by the esophageal tube and no operation was performed, and this group was treated symptomatically. Three rats from Group II died one week following esophageal and gastric perforation (25%).

Preoperatively, the animals were given 100 mg of intramuscular Rocephin (ceftriaxone sodium, Roche). All the animals were killed with an overdose (100 mg/kg) of intravenous thiopental sodium (Pentothal, Abbott) on postoperative day 30. After being sacrificed, the stomach and esophagus were totally removed. Each specimen was evaluated by the Pathology Department according to the tissue damage when compared with the control group (Table 1).

Histomorphological Evaluation

For histopathological analysis, the stomach and esophagus were totally removed and fixed in 10% neutral buffered formalin solution, and embedded in paraffin. Embedded tissues were cut into 5–µm-thick sections and stained with hematoxylin-eosin (H&E) and examined under a light microscope by

Table 1. Histopathologic parameters of the stomach and esophagus

Histopathologic parameters		Group I (n)	Group II (n)
Necrosis	Mucosa	0	1
	Submucosa	0	3
	M. propria	0	1
Ulcer		0	3
Perforation		0	3
Erosion		4	12
Hemorrhage		3	9
Inflammation	No change	0	
	Mild	2	8
	Severe	0	4

a histopathologist blinded to the study design. The histopathologic findings were scored as follows: 0: no changes; 1: minor-mild changes; and 2: severe changes according to the following parameters: 1) necrosis, 2) ulcer, 3) perforation, 4) erosion, 5) hemorrhage, and 6) inflammation.

The specimens were subsequently fixed with 10% natural buffered formalin and embedded in paraffin. Thereafter, sections were cut at a thickness of 4 µm and stained with H&E. Necrosis and depth of necrosis were evaluated according to its presence in the tissue samples.

Statistical Analysis

Statistical analysis was performed using SPSS 10.0 for Windows, and p values <0.05 were considered as statistically significant. We used Fisher's exact test for the comparison of categorical variables.

RESULTS

Acute death was observed in 5 rats immediately after administration of the corrosive agent (15%) at the beginning of the study. Three rats from Group II died due to esophageal and gastric perforation within one week (25%). Necrosis was reported in 5 non-gastrostomized rats (Figure 1); however, no necrosis was observed in the gastrostomized group (Table 1, p=0.037). No injury distal to the pylorus was observed. Normal histopathologic anatomy was observed in the control group (Figure 2). Mucosal ulcer was found (+) in 3 out of 12 specimens in Group II, while the same parameter was (-) in all specimens in Group I. Erosion was found (+) in 4 out of 12 specimens in Group I, while it was (+) in all specimens in Group II. We observed mucosal hemorrhages in 9 out of 12 specimens in Group II, but we observed only 3 mucosal hemorrhages in Group I. Inflammation was found in 2 specimens in Group I, and in all specimens in Group II

DISCUSSION

The degree of corrosive esophagus and stomach injuries depends on the form, amount and concentration of the ingested material and especially the duration of exposure. Esophageal stenosis, perforation and death can be observed after exposure to the corrosive agent. There is no standard treatment for acute corrosive esophagitis and gastritis. In mild cases, symptomatic medical treatment may be sufficient. On the other hand, in severe cases, the rates of morbidity and mortality are high. In such a condition, treatment is determined according to a "wait and see" strategy (11,12). Three

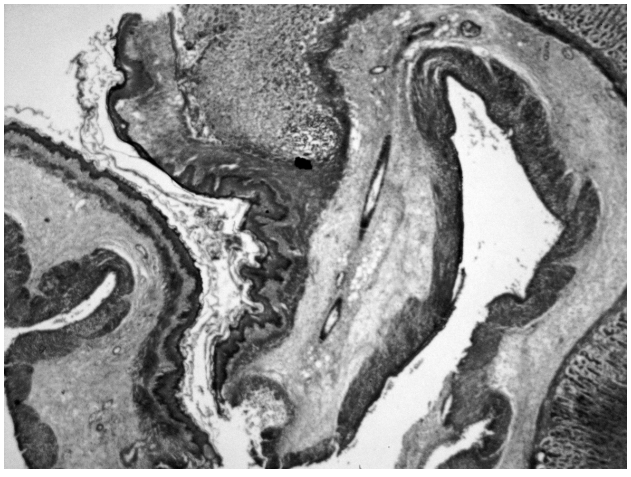

Figure 1. Necrosis and ulcer (H&E x100).

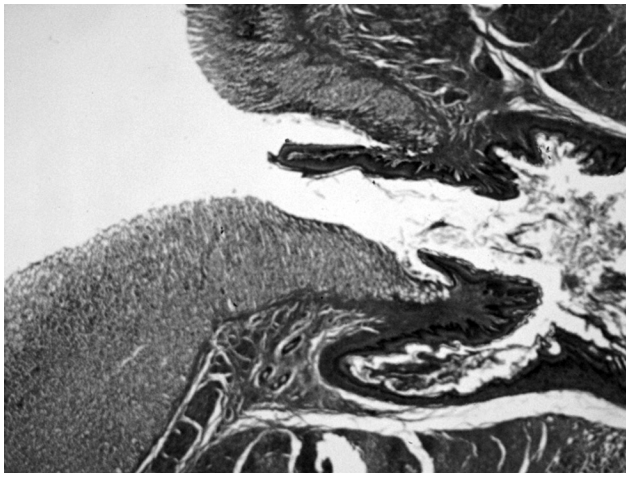

Figure 2. Normal esophagogastric line in the control group (H&E x40 and H&E x400).

rats in the non-gastrostomized group (Group II) died due to gastric perforation.

In the cases with swallowing of alkaline and acidic agents, liquefaction necrosis and coagulation necrosis are observed, respectively. Alkaline burns are observed more than acidic burns. Hydroxyl ions in the alkaline chemicals rapidly bind to free hydrogen in the tissues, and within 2–3 days cause thrombosis, tissue necrosis, bacterial infiltrations, and fatty suppurations in the tissues. Alkaline agents mainly cause burns in the esophagus, but when they reach the stomach, they can be neutralized by the acid in the stomach and lose their harmful effects to a greater extent (13). When alkaline agents are swallowed, reflex spasms occur in the esophagus and result in injury in the mediastinum. First, the mucosal injury appears followed by the muscular layer injuries. Sometimes, esophageal perforations may complicate the condition, and the agents may enter into the mediastinum, causing aortic injuries, finally resulting in death (12,14). Alkali ingestion often results in pylorospasm, with retrograde ejection of the caustic agent back up the esophagus as the stomach contracts in spasm, followed by a secondary cricopharyngeal muscle spasm, with propulsion back into the stomach. This "seesaw" motion of the caustic agent aggravates both the esophageal and the gastric burn (8,14).

According to endoscopic images, burns are graded as mild, moderate and severe. In emergent endoscopic screening, if burns are not present, the patient can be discharged from the hospital. If burns are observed, then the treatment should begin immediately. Sometimes, burning agents can cause respiratory distress, and in these cases, the airway should be kept open, and intubation or even tracheostomy may be indicated if needed. Five rats died in the early period of our study because of respiratory distress after administration of the corrosive agent. Administration of broad-spectrum antibiotics should be initiated because of the risk of esophageal-wall injuries and mediastinitis. Oral feeding, which carries risks for bacterial contamination via ingested food, should be stopped, and parenteral feeding must be started. If burns are severe and oral feeding cannot be given for a prolonged period, total parenteral nutrition should be considered or gastrostomies for feeding should be performed.

When severe corrosive burn is observed in the esophageal mucosa, endoscopic examination and nasogastric tube insertion are not carried out due to perforation risk to the mediastinum. In light of this risk, we suggest gastrostomy in these cases in the early period of burn. Despite all of these treatment modalities, the rate of stenosis in esophageal burns is as high as 20% (9). Primary treatment for these patients includes dilatation, and its frequency depends on the patients' needs. When dilatation fails, surgery is mandatory. In small segmental stenosis, resection and anastomosis may overcome this problem; however, in the case of large segmental stenosis, the stomach, small intestine, or colon transposition may be required (14). Furthermore, as is known, in cases with esophageal burns, squamous cell carcinomas may develop after 10–40 years at a rate of 3–10%, especially in the burned segments (9,12). New management methods for serious complications in acute corrosive esophagitis should be established.

In our study, we aimed to decrease the perforation risk owing to corrosive agent ingestion with immediate gastrostomy, enabling aspiration of the corrosive agent in the stomach as well as irrigation of the esophagus and stomach with physiologic saline in severe cases, since gastric tube or esophagoscopy cannot be used in a severely inflamed esophagus. When corrosive agents are swallowed, spasms occur in the cricopharyngeal muscle and gastric pyloric region. Since the lower esophageal sphincter is open, the corrosive agent flows up and down during the early stage (15). This flow worsens the already present pathologies. Thus, at this point, removal of the corrosive agents via immediate gastrostomy prevents further injury, and the harmful effects of the corrosive agents are thereby reduced. Furthermore, after corrosive agent ingestion, perforation, necrosis, erosion, hemorrhage, and inflammation were observed in the medically treated group; however, these complications, especially perforation, could be prevented in the gastrostomized group. We were unable to make any comparison of the results because there is no similar study in the literature.

In conclusion, acute severe corrosive esophagitis and gastritis are life-threatening injuries and may cause death. Their complications may last for years and unpleasant outcomes may complicate the condition of these patients. When nasogastric tube and/or esophagoscopy cannot be performed in these severe cases, immediate gastrostomy may prevent perforation by decreasing the seesaw motion and emptying the pool of caustic agent into the stomach.

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