Management of Corrosive Injury of Gastrointestinal Tract

Ruxandra Albu*, Gabriel Constantinescu+, Adriana Dumitru*, Ana-Liliana Dumitru*

Abstract

BACKGROUND: Ingestion of alkaline or acidic chemical is a common event. Patients with caustic ingestion from Bucharest and small towns near Bucharest are referred to our Hospital.

METHODS: We studied retrospectively 188 patients who ingested caustic agents between January 2003-January 2004.

We studied the type of caustic, substance, mucosal injury, systemic complications.

Caustic agents were mainly lye, strong acids (hydrochloric, sulfuric, acetic), bleach products. Initial management of these patients was to stabilize them and was focused on the immediate damage to the upper gastrointestinal tract.

Upper endoscopy was done in the first 24 hours after presentation; lesions were graded using a modification of the classification proposed by Di Constanzo et al.

Unstable patients were admitted in the intensive care unit, the mortality rate was 82%. None of them underwent surgery.

Mortality causes for these patients were perforation and catastrophic hemorrhage.

The grade of mucosal injury was predictive for the development of strictures. 24% of patients with grade 2 lesions and 92,5% of patients with grade 3 lesions developed strictures.

Treatment consisted of proton pump inhibitors, antibiotic and total parenteral nutrition.

Esophageal strictures were treated with endoscopic dilation. Only 2% were complicated. Gastric dilation was unsuccessful.

Surgical treatment consisted of gastro stomas, esophagoplasty, correction of gastric strictures.

CONCLUSIONS: Caustic ingestion is associated with a considerable morbidity and mortality despite intensive supportive care. (Revista de Medicină de Urgență, Vol. 2, Nr. 1, 29-32)

Introduction

Caustic ingestion can cause a progressive injury of the esophagus and stomach and often leads to severe morbidity and frequently death.

There are few well designed trials examining the effects of proposed specific therapy. That's the reason why the treatment outline is determined by personal experience and judgment of the physician.

Caustic agents

Caustic gastrointestinal injury can be caused by an alkaline or acidic agent. The most common cause is ingestion of strong alkali (sodium or potassium hydroxide) contained in drain cleaners, other household cleaning products, or disc batteries. The term "lye" implies substances that contain sodium or potassium hydroxide. Highly concentrated acids (hydrochloric, sulfuric, and phosphoric acid) contained in toilet bowl or swimming pool cleaners, antirust compounds, or battery fluid, are less frequently ingested. Liquid household bleach (5 percent sodium hypochlorite) ingestion is frequently reported but rarely causes severe esophageal injury [1].

Specific for our country is lye used for home made soap.

The severity and extent of esophageal and gastric damage resulting from a caustic ingestion depends upon the following factors [2]:

- the corrosive properties of the ingested substance;
- the amount, concentration and physical (solid, liquid) form of the agent;
- the surface of contact with the mucosa;
- the patients condition before the ingestion.

Physiopa thology. Alkali ingestion typically damage the esophagus more than the stomach whereas acids cause more severe gastric injury. Aspiration of either acid or alkali can also induce both larvngeal and tracheobronchial injury. [3]

Alkali-induced injury. Ingestion of alkali acutely results in a penetrating injury called liquefactive necrosis. The injury extends rapidly (within seconds) through the mucosa and wall of the esophagus towards the mediastinum until the alkali is buffered by tissue fluids. In the stomach, partial neutralization of the ingested alkali by gastric acid results in a more limited injury. Duodenal injury is much less common than the involvement of the esophagus and stomach, respectively.

Extensive transmural damage may result in esophageal, gastric, or duodenal perforation, mediastinitis, peritonitis, and death [4]. This is most likely to occur with liquid preparations which coat larger mucosal surfaces. In contrast, solid preparations generally cause oral, pharyngeal and laryngeal injury because of difficulty swallowing solid particles and local pain.

The process of liquefactive necrosis usually lasts three to four days and is associated with vascular thrombosis and mucosal inflammation, resulting in focal or extensive sloughing and ulceration. Over the ensuing two weeks, the esophageal wall becomes progressively thinner because of sloughing and the development of granulation tissue and fibrosis. Reepithelialization is usually complete one to three months later.

^{*} MD

⁺ MD, University of Medicine and Pharmacy "Carol Davila". Gastroenterology department – Bucharest Emergency Clinical Hospital. Author for correspondence; Ruxandra Albu albii@home.ro Recieved November 20, 2004, accepted April 25, 2005.

The likelihood of stricture formation depends upon the depth of damage and degree of collagen deposition [5].

Death and severe complications primarily occur in patients with severe second-degree and third-degree burns (see below) [6].

Acid-induced injury. Acid ingestion typically produces a superficial coagulation necrosis which thromboses the underlying mucosal blood vessels and consolidates the connective tissue, thereby forming a protective eschar [7]. Because acid solutions cause pain upon contact with the oropharynx, the amount of acid ingested tends to be limited. In addition, in contrast to the more viscous alkaline solutions, acid preparations tend to pass quickly into the stomach, causing less esophageal damage.

Management. The use of emetics is contraindicated because vomiting reexposes the esophagus and the oropharynx to the caustic agent aggravating the injury.

Nasogastric intubation to remove the remaining caustic material is contraindicated.

Using neutralizing agents is contraindicated because damage is generally instantaneous. Neutralization releases heat which adds thermal injury to the chemical destruction of tissue.

The patient which is asymptomatic, gives a reliable history of a low volume, accidental ingestion of low concentration acid or alkali, they are discharged and followed as outpatients.

Symptomatic patients are admitted.

Life threatening complication of injury are mediastinitis, peritonitis, respiratory distress, shock.

Clinical signs of critical patient are:

- severe injury of esophagus and stomach, dispnea, dysphagia, oral pain and odynophagia, chest pain, abdominal pain ,nausea and vomiting or hematemesis;
- impending airway obstruction: stridor, hoarseness, dysphonia, respiratory distress;
- acute peritonitis: abdominal guarding, rebound tenderness, diminished bowel sounds.

Patients and Methods

We have studied 188 patients hospitalized January 2003 –January 2004 in Emergency Hospital Bucharest for ingestion of a caustic agent.

We want to propose an algorithm of management of corrosive injury.

We studied records for 188 patients referred to our hospital last year.

Data were collected from medical records of the patients.

Types of caustic substance, mucosal injury, systemic and gastrointestinal complications, treatment and survival were scored.

Upper endoscopy was done with Olympus endoscopes-GIF Q 145 and GIF Q 160.

Evaluation of causti lesions was done by 6 well trained endoscopists.

Results

Patients were coming from Bucharest - 22%, small towns -24% and 54% from the country side.

More women (56%) than men ingested caustic substances.

Ingestion was associated with suicidal intents in 70% of cases.

Inadvertent ingestion - usually alcoholics (wrong bottle).

Caustic agents were: acids - 39% (hydrochloric, acetic, phosphoric), alkali - 40% (sodium and potassium hydrox-

ide), lye -13%, liquid household bleach and unknown - 7%. Specific for our country is lye used for home made soap.

Management

Current management was based upon our clinical experience.

33 of the patients who had ingested a small amount of a caustic substance were discharged.

The other 155 of patients were hospitalized.

For these patients first we took chest and abdominal films.

If the patient was hypotensive, he received intravenous fluids and blood products.

For patients with respiratory distress, laryngoscopy was performed in order to evaluate the need for tracheostomy.

Patients with oropharyngeal injury were monitored closely for the possible development of airway obstruction.

If the larynx was edematous endotracheal intubation was contraindicated and a tracheostomy was performed for airway control.

Endoscopy was performed as soon as possible after admission to assess the magnitude and extent of injury.

We are using modified Di Constanzo mucosal damage grading system [9]:

Grade 0 – no mucosal damage

Grade 1 – edema/erythema – see image 1

Grade 2 – moderate ulceration and/or hemorrhage - see image 2

Grade 3 – extensive ulceration and or hemorrhage - see image 3

The unstable patients were admitted in the intensive care unit.

22 of the patients were unstable in the emergency department.

18 of these patients died from complications - mainly perforation for 15 patients.

2 patients died from catastrophic upper digestive hemorrhage.

1 patient died from sepsis.

There was no surgical intervention in these patients.

The other 4 patients were transferred in the gastroenterology department.

Patients admitted in gastroenterology department -159 patients:

- 50 had grade 1 lesions;

- 82 grade 2;



Figure no. 3 Caustic esophagitis grade III

Figure no. 4 Esophageal stenosis





- the rest of 27 had grade 3 lesions.

Patients with grade 1 required no therapy. A liquid diet may be initiated and the patient may be advanced to a regular diet in 24-48 hours.

For grade 2 lesions liquids are allowed after 48 hours, the patient should receive total parenteral nutrition.

30 patients developed esophageal stenosis. The caustic substance was lye.

For grade 3 – patients are at a high risk of perforation. Proton pump inhibitor were used to reduce concomitant

acid reflux injury during the acute phase of injury.

Also, we added antibiotics to prevent a superimposed infection.

24 patients having grade 3 lesions developed gastric or esophageal stenosis.

Prognosis is variable depending upon grade of esophageal injury and the underlying medical condition of the patient. [8]

Late complications – esophageal or stomach stricture in antrum of fasting patients in midbody in those who had recently ingested food

98 patients admitted last year developed esophageal and gastric stenosis.

Esophageal strictures (see image 4) were treated with dilations to achieve and maintain an adequate lumen.

We are using Savary Guillard bougies, Olympus videoendoscope GIF Q 145 and GIF Q 160.

Patients received proton pump inhibitors and a broad spectrum antibiotic after dilation.

When the patient could swallow a semi liquid diet he was discharged. He was asked to come every week for esophageal dilation.

We tried to dilate with balloon an antral stenosis. Finally the patient was referred to surgery for distal gastric resection.

Esophageal dilations were complicated, with perforation in 2% of patients and referred to surgery.

Surgical indications for surgery *per primam* in esophageal stenosis were: technical problems for endoscopic dilation, high strictures, esophageal pseudodiverticula, suspicion or proved fistulas, patients who could not tolerate repeated dilations.

Surgical treatment consisted of gastrostomas, esophagoplasty corrective interventions for distal strictures or perforations.

Discussions

Ingestion of alkaline or acidic chemicals is a common event in our society.

Adults ingestion is deliberate – when occurred in alcoholic, psychotic, suicidal subjects; inadvertent – they ingest just a small quantity of the caustic substance

The initial management of these patients is focused on the immediate damage of the upper gastro - intestinal tract.

As soon as possible the lesions are evaluated by upper endoscopy.

Severity of mucosal damage and instability at presentation are associated with high mortality.

Treatment is targeted to systemic complications: antibiotics, correction of coagulation abnormalities and it is supportive.

Endoscopic dilations are useful only for esophageal strictures.

Gastric strictures are referred to surgery.

The lifelong complications and catastrophe of caustic injury make it one of the most challenging situations in gastroenterology.

There are few controlled studies for the management of caustic injury, that's why current management is based upon clinical experience.

Patients should undergo endoscopy once stabilized, to assess the degree of oropharyngeal, esophageal and gastric damage.

Then, patients with moderate to severe injury should be restricted for any oral intake, placed on intravenous fluids and observed for complications.

For those who require a long period without oral intake, feeding should be initiated through total parenteral nutrition.

We are not using corticosteroids, because they are not protecting against stricture formation and may produce serious side effects.

Late complications - esophageal or stomach stricture should be treated with endoscopic dilations or surgery (when endoscopy fails).

We don't know yet if using naso-enteral tube for feeding patients with severe lesions is better than total parenteral nutrition because we used it just for two patients.

We don't know either the incidence of gastric and esophageal carcinoma; until now none of our patients had this complication.

Unfortunately there are no attempts to control the presence of strong corrosives in our society.

References

1. Goldman LP, Weigert JM. Corrosive substance ingestion: a review. Am J Gastroenterol. 1984 Feb;79(2):85-90

2. Wasserman R L, Ginsburg C M. Caustic substance injuries. J Pediatrr. 1985,107: 169.

3. Kikendadall J W. Caustic ingestion injuries. Gastroenterology Clin North Am 1991,20: 847.

4. Gumaste VV, Dave P B. Ingestion of corrosive substances by adults. Am J Gastroenterology 199,287:1.

5. Johnson E E. A study of corrosive esophagitis. Laryngoscope. 1963 Dec;73:1651-96

6. Zargar A S, Kochlar R, Nagi B et al. Ingestion of corrosive alkalis. Spectrum of injury to upper gastrointestinal tract and natural history. Am. J. Gastroenterology 1992,07: 337.

7. Sarfati E, Gossot D, Assens P, Celerier M. Management of caustic ingestion in adults. Br J Surg. 1987 Feb;74(2):146-8.

8. Poley JW, Steyerberg EW, Kuipers EJ, Dees J, Hartmans R, Tilanus HW, Siersema PD. Ingestion of acid and alkaline agents: outcome and prognostic value of early upper endoscopy. Gastrointest Endosc. 2004 Sep;60(3):372-7.

9. Oakes DD, Sherck JP, Mark JB. Lye ingestion. Clinical patterns and therapeutic implications. J Thorac Cardiovasc Surg. 1982 Feb; 83(2):194-204.