therapy should not be in general encouraged, unless in infants and
toddlers where high and early dosage of corticosteroids could play
a justified role. Early endoscopic esophageal stenting by removable
SEMS, or better by biodegradable stents, could play an interesting role
in well identified patients.

Key words: Caustic; Ingestion; Endoscopy; Esophageal burns;
Toxicology; Liquitabs; Acids; Alkali; Endoscopic classification

INTRODUCTION
The ingestion of caustic substances, both accidental and voluntary,
determines a complex syndrome, characterized by severe, often irre-
versible, visceral lesions, with still remarkable mortality in highly
compromised patients. The most severe esophageal and gastric dam-
age is notoriously related to voluntary and pseudo-voluntary ingestion
of high amount or concentration of strong acids or alkali compounds.
More recently “liquitabs”, although not always strictly belonging to
the caustic category, have been recognized to cause serious damage
to the oral cavity and esophagus by rapid osmotic dehydration. Today
the ideal approach to caustic ingestion, especially in case of medium
or severe damage, should be achieved through the close cooperation
of a multidisciplinary team: emergency physician, anaesthetist, tox-
icologist, endoscopist and surgeon. After the first evaluation of symp-
toms and clinical signs, the toxicologic assessment and the radiologi-
al evaluation, upper digestive endoscopy represents the mainstay of
the diagnostic and therapeutic strategy. Despite clinical symptoms,
endoscopy is mandatory in all voluntary and pseudo-voluntary inges-
tions. Endoscopic description of the lesions must be very accurate
and possibly referred to an international well known endoscopic clas-
sification. Pictures or videos should be encouraged. After endoscopy,
eventually including EUS, the team has to decide about the need to
perform surgery (laparotomy or thoraco-laparoscopy) or to establish
a conservative therapy. In doubtful cases endoscopy can also be short
term repeated, under anaesthesiological control. Steroid treatment,
at least in the majority of cases, is unpredictable and so that kind of

© 2015 ACT. All rights reserved.

Key words: Caustic; Ingestion; Endoscopy; Esophageal burns;
Toxicology; Liquitabs; Acids; Alkali; Endoscopic classification

Rossi A. Acute Caustic Ingestion: State of Art and New Trends.
Journal of Gastroenterology and Hepatology Research 2015; 4(3):
1501-1506 Available from: URL: http://www.ghrnet.org/index.php/
jourghr/article/view/1140

INTRODUCTION
The ingestion of caustic substances, both accidental and voluntary,
determines a complex syndrome, characterized by severe, often irre-
versible, visceral lesions to which, in some cases, can be added
also systemic effects.

Mortality in highly compromised patients is still remarkable,
as well as the functional consequences following the most severe
injuries.

In the last decades we first record, quite rightly, a global increased
sensitivity to the problem which involves on the one hand the social
and health consciousness and the other, more specifically, the whole
of technical operators, such as emergency physicians, anaesthetists,
toxicologists, endoscopists and surgeons.

At the same time, industry itself is proving to be more in line with
security policies regarding the identification of caustics, achieved
through stricter compliance with the marketing rules, so that the
various products are easily recognizable thanks to adequate signals of
danger on the label.

In particular the use of safety containers, made in order to prevent
easy access by children, has significantly contributed to avoid many
of the incidental caustications.
Nevertheless, the risk of acute or chronic serious consequences is still remarkable, particularly with regard to patients which have voluntary or accidental ingestion of caustic in high quantity or high concentration.

**CAUSTIC COMPOUNDS: CHEMICAL CATEGORIES, HARMFULNESS, MODALITY OF INGESTION AND MECHANISM OF DAMAGE**

Caustics are able of causing lesions characterized by intense chemical inflammation at the walls of hollow visera, with a tendency to the necrosis which may involve all layers and lead to perforation\(^{[1-7]}\). The damage, in most severe cases, occurs within a few minutes (no later than 1 hour) after ingestion. The visceral perforation is undoubtedly the most dangerous and potentially lethal event for the patient: It can occur simultaneously in different areas of the esophagus, stomach and even the proximal small intestine, thus constituting the decisive factor for the prognosis\(^{[8-11]}\).

The severity of injury depends on a number of factors, both typical of the substance and characteristic of the patient: (1) Intrinsic corrosive power of the substance itself; (2) Concentration; (3) Type of commercial preparation (liquid, granular, paste, solid); (4) Modality of ingestion (accidental, intentional); (5) Amount of ingested; (6) State of gastric fullness (possible “self-buffering” by food).

In cases of incidental ingestion, injuries (especially in children) are often limited to the oropharynx, as the patient tends to stop swallowing and expel the substance\(^{[12-15]}\).

On the contrary a voluntary ingestion implies a forced swallowing of large amounts of caustic, with more severe consequences\(^{[1,4,5,13-17]}\).

The last consideration can be made also for the pseudo-voluntary ingestion, that is the unaware ingestion of caustic substance as result of incorrect storage, lack of indentifying tag, improper decanting in a commonly used container, such as the bottle of mineral water.

The painful reflex of the individual could act in many cases as a limiting factor against swallowing: nevertheless it must be observed as the most alkaline liquid solutions are tasteless and odorless and so they can be swallowed before protective reflexes could intervene.

Finally it should be emphasized that the vomiting, spontaneous or induced, determines a second passage (retrograde) of caustic substance into the esophagus, making the injury worse.

The vast majority of caustics cause only local effects, limited to the site of mucosal contact: exceptions to this rule are hydrofluoric acid and (partially) phosphoric acid\(^{[14]}\). Hydrofluoric acid, in particular, presents systemic toxicity mediated by its ability to bind the ionized plasmatic calcium, resulting in severe hypocalcemia: serious cardiac sequelae (ventricular fibrillation) and neuromuscular disorders (disorders of conduction) can occur.

As shown in figure 1, the three main chemical categories which can lead to caustic burns are strong acids, strong alkali and oxidating agents.

More recently, other compounds such as highly concentrated soapy detergents, particularly when trapped into water-soluble pods – the so called “liquitabs” - must be considered as very dangerous.

**Strong Acids**

Acids produce protein coagulation to which follows eschars and ulcers formation and bleeding. This coagulative necrosis could be in same way self-limited, as ulcer itself could restrict the extent of the damage. Nevertheless, in case of significant ingestion, the possibility remains theoretical and deep ulcerations or perforation may rapidly occur.

Since the esophageal mucosa is more resistant to the acid action, the stomach is the main target.

**Strong Alkali**

Alkali produce fats saponification, with colliquative necrosis, heat generation and vascular thrombotic lesions, so that the damage involves adjacent tissues.

Transmural injury with high perforation risk is the pathological consequence.

The esophagus is the main target of alkali ingestion.

**Oxidizing agents**

Although oxidizing agents do not have often extreme values on pH scale, they can be responsible for severe injuries with dehydration and necrosis both on esophageal and gastric mucosa.

**Liquid detergent capsules (“Liquitabs”)**

The last recent years have seen the emergence on the market of these soapy viscous detergents, highly concentrated and contained into a water-soluble blister (“pods”).

Although not always strictly belonging to the category of caustics, they can cause serious damage to the oral cavity and esophagus by rapid osmotic dehydration\(^{[16]}\).

This topical especially concerns children, fatefully attracted by the shape and the bright colors of liquitabs, which can be easily mistaken for candies.

**Clinical approach: A multi-disciplinary team**

Two conditions should be emphasized for the correct and current management of a patient who presents to the hospital for caustic ingestion\(^{[1,4]}\). (1) A wide collaboration of several specialists is actually mandatory. The physician of Emergency Department, the anesthetist, the toxicologist, the radiologist, the otolaryngologist, the endoscopist and the surgeon are the professionals mainly involved: their expertise, through a logical sequence of their efforts, is an essential condition to ensure the patient better chance of success; (2) Protocols of diagnosis and staging must be implemented, in order to make more “automatic” and therefore faster the whole route for the patient, especially during the first few hours after ingestion, where decisions are most critical and can affect the prognosis.
SYMPTOMS

The complex of symptoms in a patient with ingestion of caustic depends in part on injuries caused by the ingested substance, but also on the intrinsic reactivity of the individual: it is sometimes impossible to establish a precise correlation between symptoms and severity of visceral damage. Hence the need from the medical staff to carefully avoid underestimation of the problem.

SEQUENTIAL STEPS TO APPROACH THE PATIENT

1. Acceptance and assessment of vital signs: (1) Primary goal in critically ill patients is to maintain vital functions and to treat shock when present: admittance in Intensive Care Unit could be needed; (2) In any case blood tests, with particular regard to the white cell count and blood gases, for the search of possible metabolic acidosis, must be performed. Leukocytosis and metabolic acidosis are in fact biological markers of severity\(^1\); (3) The importance of not induce vomiting during patient management and not to make Gastrolusis must also be emphasized: any blindly placement of NG tube could increase the risk of perforation; (4) ECG, and following cardiological evaluation if needed, must be done.

2. Analysis of symptoms. Focus on: (1) Epigastric or abdominal pain (possible sign of perforation!); (2) Chest or substernal pain (from esophageal injury); (3) Tachypnoea (from obstructive edema of the pharyngo-esophageal junction); (4) Dysphonia, stridor, dyspnea (airway involvement); (5) Dysphagia, odynophagia; (5) Cyanosis; (6) Vomiting; (7) Hematemesis (severe injuries, extensive or deep).

3. Detection of objective signs: (1) Inspection of the oral cavity; (2) Chest or substernal pain (from esophageal injury); (3) Thoraco-abdominal assessment: search for perforation signs.

4. Identification of caustic: (1) Check the label: type of substance, commercial preparation and concentration; (2) Analysis of the pH, if possible, on a sample of substance; (3) Telephone contact with the Poison Control Center.

5. Characterization of ingestion: (1) Modality: voluntary or incidental; (2) Amount of ingested substance; (3) Spontaneous vomiting episodes (double pass into the esophagus).

6. Radiological evaluation

In the majority of cases a standard thoraco-abdominal X-ray is indicated, in order to identify signs of perforation (pneumomediastinum, pneumoperitoneum) or mediastinitis and pneumonia (including aspiration).

Moreover, the absence of these findings does not exclude a perforation or serious injury on a visceral level. Investigations more in-depth with water-soluble contrast medium (gastrografin) or by CT are therefore reserved for patients with suspected drilling in progress.

ENDOSCOPY

Endoscopy, as part of the instrumental techniques usable in the acute phase, is the mainstay of diagnostic evaluation and staging, as it allows you to check: (1) The presence of lesions; (2) The severity of lesions; (3) The extent of the lesions by considered area (for example the esophagus); (4) The topographical distribution in the upper digestive tract (from the pharynx to the duodenum); (5) The presence of objective evidences correlated to the risk of perforation.

Endoscopy should be always performed in all patients in whom the ingestion of caustic in large amount or strong concentration is sure or at least suspected, such as intentional or pseudo-voluntary one\(^1,4,5,13,20,21,22\). About 20% of them could have visceral lesions without any oral pathological finding.

On the contrary, the indication for endoscopy may be questionable in patients with accidental ingestion, especially if doubtful or poorly symptomatic, as almost always happens for children who often merely taste and lick the substance before spitting itself.

In these cases, in spite of some oral lesion, over 70% of children are free of significant visceral involvement, so that the best strategy could consist in clinical observation and endoscopy should be reserved only for selected cases\(^1,4,5,13,20,21\) (Figure 2).

Endoscopist also largely competes the indication of subsequent treatment which the patient will be submitted: ultimately, the endoscopist plays a key role in making the decision between conservative therapy and surgical intervention (either for diagnostic completion or definitely resective) For these reasons the endoscopic report must be very accurate, particularly by identifying functional or morphological characteristics that could correlate with the severity of the damage and the risk of perforation.

Hence the need to classify the lesions in endoscopic patterns reliable, reproducible, minimally exposed to subjective interpretation and especially close to real pathophysiology\(^1,4,5,6,7,13,20\).

Figure 3 (below) shows the "Niguarda '90'' classification\(^1,6,20\) which stratifies the endoscopic lesions in 4 degrees and that includes some functional findings which could indirectly reflect the parietal depth of the damage.

Although the Niguarda '90 classification is in fact suitable to any patient with caustic ingestion, regardless of the age of the subject, considering the features typically found in children we believe that for pediatric age the classification proposed by the Italian Consensus on Not Bleeding Emergency Endoscopy (AIRONE 2008), given in table 1, is more consistent\(^1,2,3\).

Timing of endoscopy

In general, the endoscopic observation must be, in a conceptual logic, as early as possible\(^1,2,3,21\). This in order to quickly select patients for which do not require additional health measures and that can be discharged, from those characterized, on the contrary, by severe or potentially evolving injuries.

However, the ideal timing can be influenced by several factors, both intrinsic to the ingested substance and related to the patient's condition, justifying sometimes a weighted delay in endoscopic evaluation. Thus, for example, emergency endoscopy is actually mandatory in critically ill patients, with clinical examination and laboratory tests suggesting a possible impending perforation. Otherwise, patients with less relevant ingestion or minor symptoms can reasonably be delayed for a few hours\(^1,2,3,20,21\).

| Table 1 Airone 2008 classification for caustic injuries in children. |
|-----------------|-------------------|-------------------|
| Grade | Endoscopic features | Extent of lesions |
|------|----------------------|-------------------|
| 0    | No lesions           |                   |
| I    | Erythema             |                   |
| II a | Pseudo-membranes     | Sectoral          |
| II b | Ulcer / necrosis     | Sectoral          |
| III a| Pseudo-membranes     | Circumferential   |
| III b| Ulcer / necrosis     | Circumferential   |
complications in the case of a "second endoscopic look" scheduled in the days following the onset of caustication.

However, we believe that, thanks to technological improvements and the pivotal anaesthesiologic care, these risks can be sufficiently contained, so that the "second-look" can and should be done at least in the following circumstances: (1) a first endoscopic examination incomplete in extent and accuracy; (2) the need for preoperative evaluation: it concerns usually patients who get worse in spite of opening endoscopy with lesions not particularly severe.

**EUS**

The preliminary study experiences of caustic-damaged mucosa by means of echoendoscopic probes or miniprobes from 12 to 20 MHz, which allow an assessment of the depth of the mucosal damage, appear positive. This interesting method however expects confirmation, especially concerning the feasibility in real urgency. Other recent works consider the simple endoscopic observation amply sufficient to address prognosis and therapy either in patients with minimal lesions and in those with severe damage.

### MANAGEMENT AND THERAPEUTIC INDICATIONS

The phase of the therapeutic measures follows the clinical and endoscopic staging. In this respect we can distinguish three basic types of strategies, carried out in relation to the three main categories of risk that are possible outcomes of staging.

A. For patients with only mild visceral lesions, without systemic involvement, is only provided medical support. The patient may be discharged with an invitation to carry out clinical and in case endoscopic remote controls.

B. Patients with severe visceral lesions, usually related to biochemical signs of systemic involvement, with the possibility of impending perforation, are routed to a timely surgical exploration, with high probability of resective therapy.

C. Patients with visceral lesions of medium severity, possibly subject to further deterioration, are undoubtedly the most problematic, and for them the Literature is still controversial. In these cases, however, it seems appropriate to suggest a careful evaluation of biochemical signs of severity and an appropriate resetting of clinical and endoscopic staging in a protected environment: in case of doubt, however, is in our opinion fully justified the need for exploratory surgery (laparoscopy, mediastinoscopy, laparotomy) in order to prevent perforation.

The type of management suggested for the category "C" from the logical point of view involves a substantial "revisiting" of the figure of the surgeon in the context of a more modern approach to the patient with caustic ingestion. In cases with endoscopically severe lesions, the surgeon is able to determine the exact state of impairment of the outermost layers of the bowel wall, filling then the limit of the intraluminal observation and gathering information clearly denied even the most experienced endoscopists.

The algorithm in figure 4 summarizes what stated above.

### MEDICAL TREATMENT

Patients with caustic ingestion should receive in acute phase adequate medical therapy, such as administration of fluids or antibiotics, depending on the severity of the clinical presentation. Literature
is instead still controversial with regard to the administration of corticosteroids, both by systemic and topical way[16-41]. Several reports are retrospective or anecdotic, but statisitcally significant demonstrations from prospective trials are still lacking. A systematic pooled analysis of fifty years of human data, published in 2007 by J. Fulton[42], fails to support the use of steroids in patients with caustic-induced grade II esophageal burns. We could conclude that today the effectivenes of steroid treatment, at least in the majority of cases, is unpredictable and so that kind of therapy should not be in general encouraged.

Nevertheless some reports, more specifically concerning infants and toddlers, seem to be favorable towards the use of high dosage of corticosteroids, starting at the early phase of caustication, so that in this particular subset of patients steroid therapy could play a justified role.

EARLY ENDOSCOPIC STENTING

Treatment of benign esophageal stenoses by removable plastic or metallic self-expandable stent (SEMS) has been developing during the last decade and now it could be considered a well established therapeutic choice, as well as endoscopic dilations.

More recently early stenting, performed just after the first endoscopic evaluation, has been encouraged with the goal of preventing the stenosis’ development, especially in case of wide and circular esophageal lesions[13,44].

Since in these cases the esophageal lumen is not yet narrow, the possible migration of the stent could represent a technical limit, that however could be overcome by anchoring the stent to a nasogastric tube. in the near future, the application of biodegradable stents, now possible migration of the stent could represent a technical limit, that since in these cases the esophageal lumen is not yet narrow, the possible migration of the stent could represent a technical limit, that however could be overcome by anchoring the stent to a nasogastric tube. in the near future, the application of biodegradable stents, now

Since in these cases the esophageal lumen is not yet narrow, the possible migration of the stent could represent a technical limit, that however could be overcome by anchoring the stent to a nasogastric tube. in the near future, the application of biodegradable stents, now

CONFLICT OF INTERESTS

The authors declare that they have no conflict of interests.

REFERENCES

1 Alfredo Rossi. Consensus statement sulle urgenze endoscopiche non emorragiche in età pediatrica. Ed Area Qualità, Milano. 2008.
2 Alfredo Rossi. Ingestione di sostanze caustiche: come affrontarla nell’attuale realtà sanitaria? Giorn Ital End Dig 2004; 27: 9-13
3 Andreoni B, Marini A, Gavennini M, Biffi R, Tiberio G, Farina ML, Rossi A. Emergency management of caustic ingestion in adults. Surg Today 1995; 25(2): 119-124
4 Claudio Grosso. Ingestione di sostanze caustiche: inquadramento clinico ed endoscopia in urgenza nel paziente adulto. Giorn Ital End Dig 2004; 27: 45-53
5 Friedman EM. Caustic ingestions and foreign body aspirations: an overlooked form of child abuse. Ann Otol Rhinol Laryngol 1987 Nov-Dec; 96(6): 709-712
6 Kay M and Wyllie R. Caustic ingestion in children. Current Opinion in Pediatrics 2009; 21: 651-654.
7 Manes G, Rossi A. L’ingestione di sostanze caustiche: guida la management del paziente acuto. Giorn Ital End Dig 2012; 38: 23-27
8 Alinejad A. Caustic Injury to the Upper Gastrointestinal Tract. Acta Paediatr 1995; 84(10): 1177-1182
9 Chiu HM, Lin JT, Huang SP, Chen CH, Yang CS, Wang HP. Prediction of bleeding and stricture formation after corrosive ingestion by EUS concurrent with upper endoscopy. Gastrointest Endosc 2004; 60(5): 827-833
10 Chou SH, Chang YT, Li HP, Huang MF, Lee CH, Lee KW. Factors predicting hospital mortality of patients with corrosive gastrointestinal injuries receiving esophagogastrectomy in the acute stage. World J Surg 2010; 34: 2383-2388
11 Christesen HB. Prediction of complications following unintentional caustic ingestion in children. Is endoscopy always necessary? Acta Paediatr 1995 Oct; 84(10):1177-1182
12 Betalli F, Balchetti D, Giuliani S, Pane A, Dall’Oglio L, de Angelis GL, Caldore M, Romano C, Gamba P, Balvo V. Caustic ingestion in children: is endoscopy always indicated? The results of an Italian multicenter observational study. Gastrointest Endosc 2008; 68(3): 434-439
13 Conlini S, Swaray-Deen A and Scarpignato C. Caustic ingestion in children: is endoscopy always indicated? A perspective from a Sierra Leone experience. Gastrointest Endosc 2009; 69(6): 1191-1192
14 Denney W, Ahmad N, Dillard B, Nowicki MJ. Children Will Eat the Strangest Things. A 10-Year Retrospective Analysis of Foreign Body and Caustic Ingestions From a Single Academic Center. Pediatr Emerg Care 2012; 28: 731-734
15 Marta Bini. Ingestione di caustici in età pediatrica. Giorn Ital End Dig 2004; 27: 57-62
16 Cellerier M. Prise en charge des aesophagites caustiques chez l’adulte. Anu Chir 1996; 50(6): 449-455
17 Mas E, Breton A, Lachaux A. Prise en charge des enfants après ingestion de substances acides ou alcalines. Archives de Pédiatrie 2012; 19(12): 1362-1368
18 MMWR (Morbidity and Mortality Weekly Report). Health hazards associated with laundry detergent pods - United States May-June. 2012. 61 (41)
19 Cheng YJ, Kao EL. Arterial blood gas analysis in acute caustic ingestion injuries. Surg Today 2003; 33(7): 485-485
20 Poleyn 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): 372-377
21 Rigo Gp, Camellini L, Azzolini F, Guazzetti S, Bedogni G, Meriggi A, Bellis L, Scarcelli A, Manenti F. What is the Utility of Selected Clinical and Endoscopic Parameters in Predicting the Risk of Death after Caustic Ingestion. Endoscopy 2002; 34(4): 304-310
22 Schaffer SB, Hebert AF. Caustic ingestion. J La State Med Soc 2000 Dec; 152(12): 590-596
23 Hawking DB, Demeter MJ, Barnett TE. Caustic ingestion: controversies in management. A review of 214 cases. Laryn-
© 2015 ACT. All rights reserved.

Peer reviewers: Jun Kobayashi, MD, PhD, Professor, Laboratory of Pathophysiology, Department of Clinical Dietetics and Human Nutrition, Faculty of Pharmaceutical Science, Josai University, 1-1 Keyakidai, Sakado, Saitama, 350-0295, Japan;

Rossi A et al. Acute caustic ingestion

goscope 90:98. 1980
24 Temiz A, Oguzkurt P, Ezer SS, Inci E, Hicsonmez A. Predictability of outcome of caustic ingestion by esophagogastrroduodenoscopy in children. World J Gastroenterol 2012; 18(10): 1098-1103
25 Lamireau T, Rebeuissoux L, Denis D Lancelin F, Vergnes P, Fayon M. Accidental caustic ingestion in children: is endoscopy always mandatory? JPGN 2001; 33: 81-84
26 Arcidiacono R, Rossi A, Grosso C, Bini M, Gambitta P, Zanasi G. Proposition d’une nouvelle classification endoscopique des lesions par ingestion de caustiques. Acta Endosc 1992; 22(4): 413-418
27 Tognini L, Gavolini M, Scacchi GL, Martino E, Palladino P, Belloli F, Tiberio GA, Massari M, Tiberio G. Indicazioni alla chirurgia d’urgenza nelle lesioni da ingestione di caustici.RUolo di una classificazione endoscopica originale. Chir Ital 1998; 50(5-6): 41-46
28 Zargar SA, Kochhar R, Mehta S, Mehta SK. The role of fiberoptic endoscopy in the management of corrosive ingestion and modified endoscopic classification of burns. Gastrointest Endosc 1991; 37: 165-169
29 Boyce HW. Medication-Induced Esophagitis and Caustic Ingestion. In: Katz PO, Devault KR, Hinder RA (eds). GI in the next century. 1999 AGA Postgraduate Spring Course, Syllabus. Orlando: American Gastroenterology Association 1999: 207-216
30 Byrne WJ. Foreign Bodies, bezoars and caustic ingestion. Gastrointestinal Endoscopy Clin of North Am 1994; 1: 99-119
31 Chien WC, Pai L, Lin CC, Chen HC. Epidemiology of hospitalized burns patients in Taiwan. Burns 2003 Sep; 29(6): 582-588
32 Bernhardt J, Ptok H, Wilhelm L, Ludwig K. Caustic acid burn of the upper gastrointestinal tract: first use of endosonography to evaluate the severity of the injury. Surg Endosc 2002 Jun; 16(6): 1004
33 Kamijo Y, Kondo I, Kokuto M, Kataoka Y, Soma K. Miniprobe ultrasonography for determining prognosis in corrosive esophagitis. Am J Gastroenterol 2004 May; 99(5): 851-854
34 Kamijo Y, Kondo I, Soma K, Imaizumi H, Ohwada T. Alkaline esophagitis evaluated by endoscopic ultrasound. J Toxicol Clin Toxicol 2001; 39(6): 623-625
35 Huscher CG, Mingoli A, Merue A, Sgarzini G. Laparoscopy can be very effective in reducing mortality rate for caustic ingestion in suicide attempt. World J Surg 2011; 35: 2363-2364
36 Anderson KD, Rouse TM, Randolph JG. A controlled trial of corticosteroids in children with corrosive injury of the esophagus. N Engl J Med 1990 Sep 6; 323(10): 637-640
37 Boukthir S, Feti I, Mrad SM, Mongaliti MA, Debbabi A, Barsaoui S. High doses of steroids in the management of caustic esophageal burns in children. Arch Pediatr 2004 Jan; 11(1): 13-7
38 Camargo MA, Lopes LR, Grangeia T de A, Andreollo NA, Brandalise NA. Use of corticosteroids after esophageal dilatation on patients with corrosive stenosis: prospective, randomized and double-blind study. Rev Assoc Med Bras 2003 Jul-Sep; 49(3): 286-292
39 Howell JM, Dalsey WC, Hartsell FW, Butzin CA. Steroids for the treatment of corrosive esophageal injury: a statistical analysis of past studies. Am J Emerg Med 1992 Sep; 10(5): 421-425
40 Pelkova D, Navratil D. Do corticosteroids prevent oesophageal stricture after corrosive ingestion? Toxicol Rev 2005; 24(2): 125-129
41 Bautista A, Varela R, Villanueva A, Estevez E, Tojo R, Cadranel S. Effects of prednisolone and dexamethasone in children with alkali burns of the oesophagus. Eur J Pediatr Surg 1996 Aug; 6(4): 198-203
42 Fulton JA, Hoffman RS. Steroids in second degree caustic burns of the esophagus: a systematic pooled analysis of fifty years of human data: 1956-2006. Clinical Toxicology 2007; 45: 402-408
43 Conio M, Bianchi S e De Ceglie A. Protezi rimuovibili e riassorbibili nel trattamento delle stenosi benigne esofagee. Giorn Ital End Dig 2012; 35: 245-249
44 Manta R, Conigliaro R, Bertani H, Manno M, Soliman A, Fedeli P, Bassotti G. Self-Expandable Metal Stenting of Refractory Upper Gut Corrosive Strictures: A New Role for Endoscopy? Case Reports in Gastrointestinal Medicine 2011, Article ID 346413
45 Bychkova OV and Lazyuk I. Bio-degradable stents—a new approach to the treatment of caustic stenoses in children. Folia Gastroenterol Hepatol 2009; 7(1): 1-6

© 2015 ACT. All rights reserved.