Ingestion of acid and alkaline agents: outcome and prognostic value of early upper endoscopy

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Background: **Ingestion of caustic substances often leads to severe morbidity and, frequently, death. This study compared complications and survival for patients who ingested an acidic substance, mainly glacial acetic acid, or an alkaline agent.**

Methods: **Records for 179 patients hospitalized for ingestion of a caustic agent (85 acid [75 glacial acetic acid], 94 alkali) were reviewed. Mucosal injury, systemic and GI complications, and survival were scored.**

Results: **Outcome was less favorable for patients who ingested acid compared with those who ingested alkali with respect to mucosal injury (median: grade 2 vs. grade 1; $p = 0.013$), hospital stay (mean: 9.9 vs. 7.2 days; $p = 0.01$), admittance to the intensive care unit (44% vs. 22%; $p = 0.002$), systemic complications (24% vs. 3%; $p < 0.001$), perforation (6% vs. 0%; $p = 0.017$), and mortality (14% vs. 2%; $p = 0.003$). There was no difference in the development of strictures (acid, 15% vs. alkali, 17%). The grade of mucosal injury at endoscopy was the strongest predictive factor for the occurrence of systemic and GI complications and mortality (relative risk 9: 95% CI[3, 30]). Ten of 29 (34%) patients with strictures were treated by endoscopic dilation alone, whereas the others primarily (n = 7) or secondarily (n = 11) underwent surgery. One patient with an esophageal stricture died from systemic complications.**

Conclusions: **Acid ingestion, particularly glacial acetic acid, is associated with a higher frequency of complications and mortality rate than alkali ingestion. Early endoscopy probably is safe and provides important prognostic information. Endoscopic treatment of caustic-induced strictures is only moderately successful. (Gastrointest Endosc 2004;60:372-7.)**

Inadvertent or deliberate ingestion of alkaline and acidic agents is relatively common and often leads to severe morbidity and sometimes death. Despite the increasing use of "child proof" packages and containers for household caustic agents, accidental ingestion by children still occurs with alarming frequency.[1] Among adults, most ingestions of caustic substances are intentional, i.e., for the purpose of suicide. Chemical differences between alkaline and acid agents are probably not important in clinical practice because both strong acids and alkaline chemicals penetrate tissue extremely rapidly to cause full thickness damage to the GI wall.[2,3] In certain ethnic populations, in the Netherlands, particularly people of Surinam of Hindustan origin, ingestion with glacial acetic acid (GAA), also known as concentrated (80%) acetic acid, is a relatively common mode of attempted suicide. However, not every ingestion of GAA is intentional; GAA is used in the Hindustan-Surinam kitchen for preparing food, and, thus, accidental ingestion occurs as well.[4] Apart from severe damage to the mucosa and even the deeper layers of the upper-GI tract, which may result in perforation or stricture formation, ingestion of GAA also results in systemic complications, i.e., renal and hepatic insufficiency, hemolysis, and diffuse intravascular coagulation.[5]

Other acid chemicals (e.g., sulfuric acid, hydrochloric acid) and alkaline agents (e.g., caustic soda [NaOH]) may also damage the esophagus, the stomach, and the duodenum. However, unlike GAA, ingestion of these substances usually does not result in systemic complications.

Several series of cases of caustic ingestion have been published over the last 3 decades, but these mainly emphasize the ingestion of caustic soda and/or the outcome of caustic ingestion in children.[6-12] There are few data on the outcome of acid or alkaline

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Table 1. Modified Di Costanzo mucosal damage grading system

Grade 0	No endoscopic damage
Grade 1	Edema, erythema, and/or exudate
Grade 2	Moderate ulceration and/or hemorrhage
Grade 3	Extensive ulceration and/or hemorrhage

ingestion by adults or the prognostic value of early upper endoscopy in these patients. Thus, a study of all patients admitted to our unit between 1980 and 2002 after ingestion of a caustic agent was conducted. Complications and survival for patients who ingested an acid (mainly GAA) were compared with those of patients who ingested an alkaline substance. The prognostic value of early endoscopy and the results of dilation of strictures caused by caustic agents were also studied.

PATIENTS AND METHODS

Patients

Medical records were reviewed for 179 adolescent and adult patients admitted to our unit from 1980 to 2002 after ingestion of a caustic agent. One hundred sixty-one (90%) patients presented within 2 to 24 hours after the ingestion, 10 presented between 24 and 48 hours thereafter, and 8 were transferred from other hospitals at more than 48 hours after ingestion. The reason for transfer of the latter 8 patients was the severity of the clinical condition caused by GI or systemic complications; these patients were included only in the analysis of long-term outcomes. All patients underwent endoscopy within 8 hours of admission. Of patients who ingested an acid, more than 85% drank GAA. A variety of alkaline substances were ingested, such as household cleaners, bleach products, and detergents.

Methods

Endoscopic intubation of the endoscope was performed under direct vision after an initial inspection of the hypopharynx. During endoscopy, air insufflation and retroflexion were carefully performed to avoid iatrogenic damage. In the majority of patients, endoscopy was performed without sedation to minimize the risk of aspiration. Mucosal damage was graded by using a modification of the classification proposed by Di Costanzo et al.[13] (Table 1 and Fig. 1). A number 10-Charrière nasogastric or nasoduodenal tube was routinely placed for the purpose of feeding the patient. Endoscopy was repeated only when indicated, usually because of the development of symptoms suggesting stricture formation or for replacement of the feeding tube. After discharge from the hospital, patients were regularly seen in the outpatient clinic for at least 1 year. When necessary, follow-up information was obtained by contacting the patient or the primary care physician.

Outcome parameters assessed were the following: systemic complications (in particular, renal insufficiency, liver damage, diffuse intravascular coagulation, and hemolysis), upper-GI complications (in particular, perforation and stricture formation), and mortality. Liver damage was defined as an elevation in the serum level of alanine aminotransferase and aspartate aminotransferase of more than 3 times the upper normal limit. Renal insufficiency was defined as a plasma creatinine level of greater than 150 μmol/L (normal: <110 μmol/L) on at least 2 consecutive days. Standard criteria were used for the diagnoses of diffuse intravascular coagulation and/or hemolysis: decreased platelet count; prolonged plasma coagulation time; decreased antithrombin or fibrinogen levels; and an elevated serum level of lactic dehydrogenase, with a low plasma haptoglobin concentration. Perforation was diagnosed by typical findings on plain x-ray films of the abdomen.

Table 2. Clinical characteristics

Patients	179
Women (%)	99 (55%)
Median age, y (range)	27 (12-81)
Ingested agent (%)	
Concentrated (80%) acetic acid	75 (42%)
Other acids	10 (6%)
Alkali	94 (52%)
Reason for ingestion (%)	
Suicide	153 (85%)
Accident	26 (15%)

A significant stricture was considered to be present if a patient developed dysphagia, regurgitation, or difficulty in swallowing, and a stricture was evident at endoscopy through which it was not possible to pass a standard 9.5-mm–diameter endoscope (Q100/200; Olympus Optical Europe, Hamburg, Germany). Subsequent to the diagnosis of a stricture, dilation usually was performed by using polyvinyl over-the-guidewire dilators (Savary-Guillard; Wilson-Cook, Salem, N.C.) on an outpatient basis, with the patient under conscious sedation. Stepwise dilation was performed, if necessary in multiple sessions, until an 18-mm diameter dilator could be passed. The decision to perform surgery for stricture management was made in a multidisciplinary setting by consensus of all physicians involved.

Statistical analysis

Data were analyzed by using a statistical software package (SPSS, version 10.0; SPSS Inc. Headquarters, 233 S. Wacker Drive, 11th floor, Chicago, Ill.). Differences between groups were analyzed by using a chi-square test for categorical variables and Mann-Whitney *U* test for continuous variables. Logistic regression analysis was used to estimate odds ratios, which were interpreted as relative risk (RR).

RESULTS

Baseline characteristics for all patients are summarized in Table 2. In total, 153 of the 179 (85%) patients ingested the caustic substance with the intention of suicide.

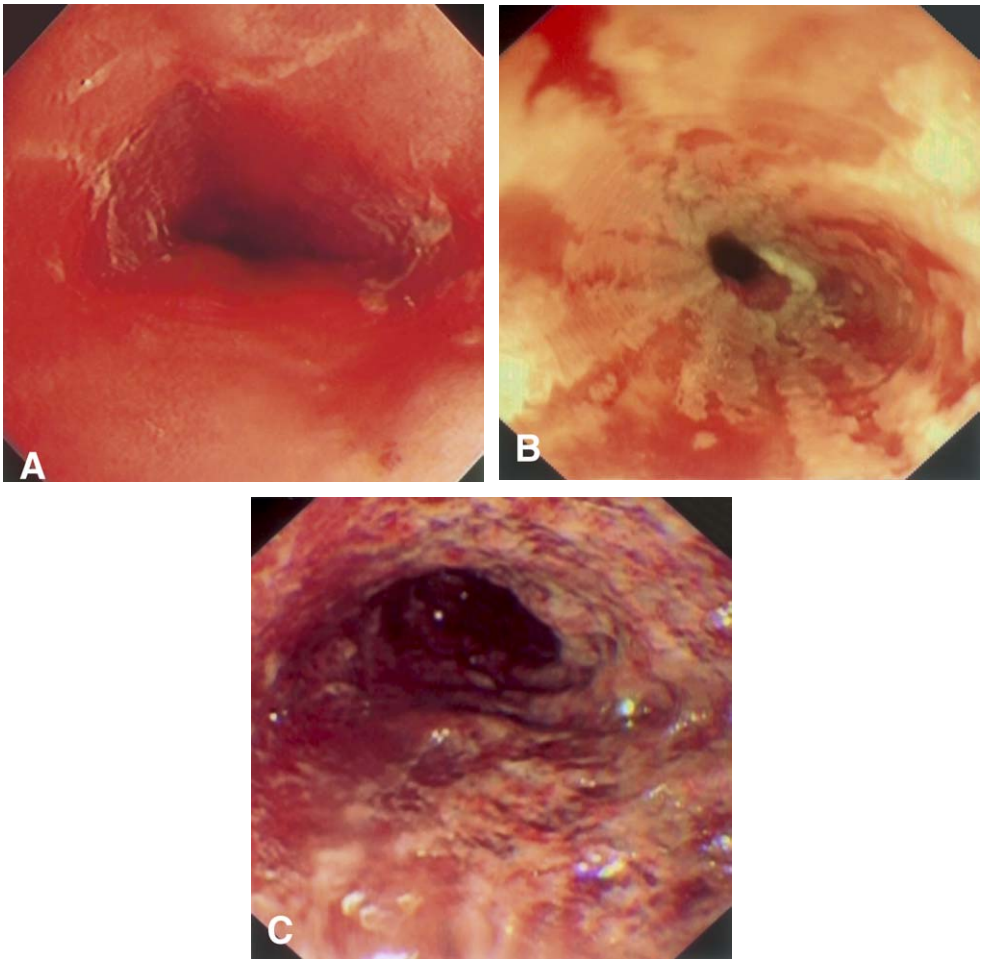

Figure 1. A. Endoscopic view showing grade 1 damage (diffuse erythema, superficial exudate) of esophageal mucosa caused by lye ingestion. **B,** Endoscopic view showing grade 2 (moderate ulceration) damage of esophageal mucosa after ingestion of glacial acetic acid. **C,** Endoscopic view showing grade 3 damage (severe, deep ulceration, and spontaneous hemorrhage) of esophageal mucosa after ingestion of glacial acetic acid.

Patients who ingested an acid were hospitalized for a longer period of time ($p = 0.01$) and were more likely to be admitted to an intensive care unit ($p = 0.002$), than patients who ingested alkali (Table 3).

Systemic complications occurred more frequently among patients who ingested an acid (20/85, 24%) than those who drank an alkaline substance (3/94, 3%; $p < 0.001$) (Table 3). The majority of patients with a systemic complication developed more than one systemic complication.

Mucosal damage to the upper-GI tract was more severe in the acid group compared with the alkali group: median grade 2 vs. grade 1 ($p = 0.013$) (Table 4). More patients in the acid group had at least grade 2 mucosal damage (60/85 [71%] vs. 46/94 [49%]). The overall mortality rate was high (14/179), especially in the acid group (14% vs. 2%; $p = 0.003$) (Table 3). Most patients died as a consequence of systemic complications, i.e., intractable bleeding caused by diffuse intravascular coagulation and hemolysis, multiorgan failure, sepsis, or a combination of these complications. The majority of deaths occurred within 24 to 48 hours after admission to the intensive care unit. For 3 patients, death was caused by a perforation.

The esophagus was the most severely affected segment of the upper-GI tract in most patients (n = 141, 79%), although damage to the stomach, especially the fundus and the body, was relatively common (n = 91, 51%). The duodenum was affected only occasionally (n = 11, 6%), and in no patient was the damage more severe than in the stomach or the esophagus. The frequency of stricture formation did not differ between the two groups; perforation occurred only in the acid group (Table 3).

Strictures (29 patients, 32 strictures) developed most commonly at 6 to 12 weeks after ingestion of the

Table 3. Outcome and survival for 179 patients with caustic ingestion

	Acid (n = 85)	Alkali (n = 94)	*p* Value
Mean hospital stay, d	9.9	7.2	0.01
Admittance to ICU	37 (44%)	21 (22%)	0.002
Systemic complications			
Renal insufficiency*	12 (14%)	1 (1%)	
Hepatic dysfunction†	12 (14%)	2 (2%)	
DIC	10 (12%)	2 (2%)	
Hemolysis	14 (16%)	1 (1%)	
Total	20 (24%)	3 (3%)	<0.001
GI complications			
Perforation	6 (7%)	0	0.017
Stricture	13 (15%)	16 (17%)	0.75
Fistula	0	1 (1%)	
Mortality			
From systemic complications	9 (11%)	1 (1%)	
From GI complications	3 (3%)	1 (1%)	
Overall mortality	12 (14%)	2 (2%)	0.003

ICU, Intensive care unit; *DIC*, diffuse intravascular coagulation.
*Creatinine > 150 μmol/L.
†Alanine aminotransferase T > 3 × upper limit of normal.

caustic substance (Fig. 2). One patient with a stricture died of systemic complications before dilation could be performed. The others were treated by repeated dilation or underwent surgery. Most strictures were in the esophagus (n = 25); a few occurred in the stomach (n = 5), but stricturing of the duodenum was rare (n = 2). In some patients, there was more than one stricture. Dilation was the initial treatment modality for 21 (72%) of the 29 patients. Seven patients with strictures (24%) underwent primary surgery when the chances for success of endoscopic treatment was considered to be low because of either the length or the number of strictures (Fig. 2). Of the 21 endoscopically treated patients, 10 had dilation alone (34%) and 11 were secondarily treated by surgery (38%) because of unsuccessful endoscopic treatment. The median number dilation sessions was 6 (range 1-84). Dilation was safe; no perforation occurred.

Logistic regression analysis showed that the severity of mucosal damage was the most predominant predictive factor for the occurrence of death and systemic complications. The RR for death or systemic complications was 9: 95% CI[3, 30], $p < 0.001$), which means that with each incremental increase in the endoscopic mucosal damage grade, the risk increased by a factor of 9. The degree of mucosal damage was more important for predicting outcome than the type of substance ingested. The RR for developing a GI or systemic complication after acid ingestion was 3: 95% CI[0.3, 15], p = not significant), after adjusting for the severity of mucosal damage.

Table 4. Endoscopic grading of mucosal damage

	Endoscopic grade (%)			
Agent (%)	0	1	2	3
GAA (n = 85)	8 (9)	17 (20)	32 (38)	28 (33)
Alkali (n = 94)	21 (22)	27 (29)	21 (22)	25 (27)
Total (n = 179)	29 (16)	44 (24)	53 (30)	53 (30)

GAA, Glacial acetic acid.
Median score GAA = 2; median score alkali = 1; $p = 0.013$ for difference in distribution.

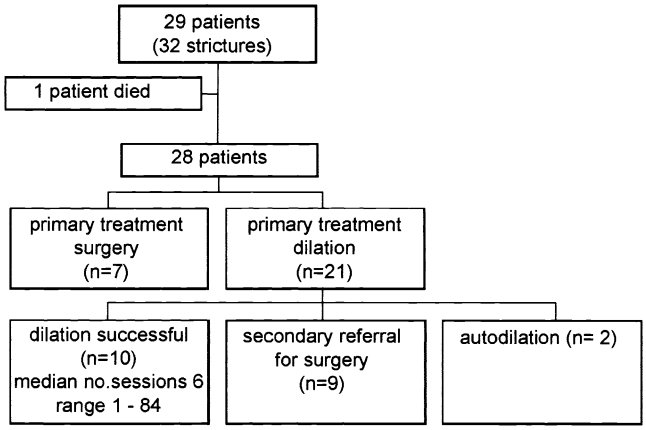

Figure 2. Flow chart for patients who developed strictures after caustic ingestion. One patient had no specific treatment and died from systemic complications.

DISCUSSION

Ingestion of alkaline or acidic chemicals is a common event, both in children and adults. In children, most such events are inadvertent; in adults, ingestion usually is a deliberate attempt to commit suicide. In the latter group, the incidence of caustic ingestion and the substances ingested may differ between populations, and between men and women. The initial management of these patients often is focused on the immediate damage to the upper-GI tract, which usually is evaluated by upper endoscopy within the first 24 hours after presentation. Nevertheless, data are scarce with regard to the long-term outcome, the relation between outcome and the type of substance ingested, and the value of early endoscopy.

The results of the present study show that ingestion of acidic or alkaline agents frequently is associated with significant morbidity and mortality, which are worse with ingestion of an acidic compared with an alkaline agent (mortality 14% vs. 2%). In other series of patients who ingested caustic

substances, mortality rates usually were well below 5%.[7-9,14,15] There are, however, several differences between the present and prior series.

The lower mortality in series of pediatric patients might be explained by ingestion of smaller amounts of the chemical agent.[10,14-16] However, stricture formation in pediatric patients is relatively common; in one study, it was 35% of the patients.[14] Another reason for the relatively high mortality and morbidity rates in the present series might be the severity of the mucosal damage; 59% of all patients had at least moderate ulceration and/or hemorrhage (Di Costanzo grade 2) in the upper-GI tract (Table 4). Mucosal damage was even more pronounced in the group that ingested acid; almost three quarters had moderate or severe mucosal damage at endoscopy. In addition, other series included mainly patients who ingested alkaline agents.[9,15,17] Only a few studies include significant numbers of patients who ingested strong acids.[3,18,19] In these series, mortality rates for patients who ingested a strong acid were as high,[3] or even higher,[19] compared with the mortality rate for the acid group in the current study.

There are few publications concerning ingestion of GAA, and these are either old[20] or merely case reports.[4,5] GAA is extremely aggressive with respect to tissue; mucosal necrosis can occur even when the ingested volume is small. Apart from this, the most distinctive feature of GAA is its ability to cause systemic complications. Although all strong acids can cause some degree of metabolic acidosis when ingested, and sometimes even mild hemolysis, the capacity of GAA to frequently cause severe systemic complications is remarkable. It has been suggested that the systemic complications are, at least partly, caused by a direct cytotoxic effect of GAA on erythrocytes after absorption.[20] In the present series, systemic complications occurred in 24% of all patients who ingested acid (mainly GAA), and 45% (9/20) of those with one or more systemic complication died. Treatment of systemic complications is supportive, i.e., dialysis, transfusion, and correction of coagulation abnormalities, if possible.

There was a significant difference in the occurrence of perforation between patients who ingested an acid vs. an alkaline substance. In the studies mentioned above,[13-15,17,21] perforation occurred only occasionally after alkali/lye ingestion. In the current study, perforation occurred only in the acid group and only when severe mucosal damage was evident at endoscopy. These patients usually underwent emergency surgery if permitted by their clinical condition. Despite surgical intervention, 3 of 6 (50%) patients with a perforation caused by acid ingestion died.

There was no difference in the present study between alkali vs. acid ingestion with respect to the occurrence of upper-GI strictures (acid [15%] vs. alkali [17%]). In prior studies of stricture formation after caustic ingestion, the frequency varied between 5% and 75%.[14,15,17,22] These differences may be explained by variations in the definition of a stricture. For studies in which radiologic criteria were used, the frequency of stricture formation usually is higher compared with that found in studies in which clinical criteria were used, as in the present study. Published data regarding treatment with corticosteroids are conflicting.[6,8,9,14,15,23] In our opinion, there is no place for the routine use of corticosteroids in the treatment of caustic ingestion, because the efficacy of this therapy is not proven. In addition, there is a possible association between administration of corticosteroids and increased risks of perforation and infection.[15] The role for prophylactic administration of antibiotics requires further study.

Only a third of the patients with strictures in the current study were managed by endoscopic means alone. A similar result was observed in a study of patients with gastric outlet obstruction caused by ingestion of caustic substances who were treated by balloon dilation.[24] The reason for the poor response to dilation is unclear. In our experience, symptoms of dysphagia frequently recur within a few days after dilation. It might be that the depth of injury caused by the caustic agent results in transmural fibrosis and, thereby, a more rigid, non-compliant esophageal wall. Histopathologic assessment of 3 esophageal resection specimens from patients with persistent strictures despite repeated dilation confirmed the presence of extensive fibrosis through all layers of the esophageal wall (data not shown). Because the burden of repeated endoscopic procedures can be high for patients, especially because multiple dilation sessions are needed in the majority (Fig. 2),[25] it seems prudent to consider surgery, even during the early stages of treatment. An alternative endoscopic approach might be to treat short strictures by electrosurgical needle-knife incision, although there is almost no reported experience with this technique in this setting.[26]

One of the most challenging aspects of the management of patients who ingest caustic agents is to predict which of them are at highest risk for systemic and/or GI complications. Based on a retrospective study, a risk score system was postulated in which age, ingestion of a strong acid, leukocytosis, and gastric ulceration or necrosis were used to estimate the probability of survival.[3] Another study, in which only ingestion of alkaline chemicals was considered, suggested that the amount of the

substance ingested is the most important factor in predicting stricture formation.[15] In actual practice, however, it often is difficult to accurately estimate this because patients often are unconscious or uncooperative. Even if they are not, estimates of the volume of one or more swallows are unlikely to be accurate.

Despite the need for caution when conclusions are based on retrospective data, the present study shows that early endoscopy is safe (no complication noted) and useful for estimating the severity of morbidity for patients who accidentally or intentionally ingest a corrosive substance. If mucosal damage is absent or is only mild (Di Costanzo grade 0 or 1), the risk of significant morbidity and death is negligible and further follow-up or observation is not indicated. However, patients with moderate to severe mucosal damage (grade 2 or 3) are at increased risk for serious morbidity and even death, and intensive treatment and follow-up are warranted.

In conclusion, despite intensive supportive care and early surgical intervention when indicated, ingestion of a caustic substance is associated with considerable morbidity and mortality. In particular, patients who ingest GAA have a poor prognosis, especially if mucosal damage is severe. Endoscopic treatment of caustic-induced strictures is successful only in a minority of patients, and elective surgery should be considered for these patients at an early stage.

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