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What is the Utility of Selected Clinical and Endoscopic Parameters in Predicting the Risk of Death after Caustic Ingestion?

Background and Study Aims: Despite the increasing use of early esophagogastroduodenoscopy, the prognostic evaluation and triage of patients who have ingested caustic material is challenging. We evaluated the usefulness of selected clinical and endoscopic parameters in predicting the risk of death after ingestion of caustic substances.

Patients and Methods: Clinical and endoscopic parameters were obtained from the records of all the patients admitted to our endoscopy unit because of ingestion of caustic material between 1 March 1982 and 30 June 1999. Parameters significantly associated with the risk of death by univariate analysis were entered into a multivariate logistic model. The independent predictors of death by multivariate analysis were used to build a risk score system.

Results: Out of 210 patients, 13 underwent emergency surgery (6.2%) and 25 died (11.9%). Multivariate analysis identified the following as independent predictors of death: age (10-year intervals; odds ratio [OR] 2.4; 95% confidence interval 1.4–4.1), ingestion of strong acids (OR 7.9; 1.8–35.3), white blood cell count at admission ≥ 20 000 units/mm^3 (OR 6.0; 1.3–28), deep gastric ulcers (OR 9.7; 1.4–66.8), and gastric necrosis (OR 20.9; 4.7–91.8). The values of the risk score system devised from the results of the multivariate analysis ranged from 1 to 16. No patient scoring < 10 points died and just one of the patients scoring > 14 points survived.

Conclusion: Age, ingestion of a strong acid, leucocytosis, deep gastric ulcers, and gastric necrosis are predictive of death after caustic ingestion. A risk score system including these predictors may be useful in prognostic evaluation.

Introduction

In Italy, four out of 100 000 hospital admissions yearly are due to caustic ingestion [1]. The clinical management of patients with caustic ingestion is difficult because of the lack of clear relationships between symptoms and severity of injury to the upper gastrointestinal tract [2–4]. Moreover the injury often extends to the upper and lower airways and may trigger a systemic inflammatory response [5]. Symptoms may develop rapidly or be delayed [6]. Early esophagogastroduodenoscopy (EGD) allows a rapid, safe and accurate assessment of the location, extent and severity of damage to the upper gastrointestinal tract [7–9] and is the diagnostic procedure of choice. Endoscopic findings correlate with clinical outcome [7,9–12]. However, it seems reasonable to suppose that a better assessment of the risk of death after caustic ingestion could be achieved by integrating clinical and endoscopic data. To test this assumption we performed a retrospective study aimed at identifying clinical and endoscopic parameters which could predict the risk of death after caustic ingestion.

Patients and Methods

Patients

All the patients at our unit between 1 March 1982 and 30 June 1999, who underwent EGD because of caustic ingestion, were retrospectively studied. We excluded from the analysis any pa-

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tients admitted to the emergency department later than 12 h following caustic ingestion and patients with overt perforation, who did not undergo EGD. Perforation was confirmed by plain abdominal films obtained on admission which revealed free air. EGD was always performed within 24 h following caustic ingestion. Published recommendations for the conduct of EGD in patients with caustic ingestion were followed [7,9]: the endoscope was introduced under visual control, insufflation was gentle, retrovisual methods were performed carefully or avoided if the stomach was severely injured.

Selection of Clinical and Endoscopic Predictors

Deaths occurring during hospital stay represented the outcome of interest. Causes of death were obtained from death certificates.

After a search of the relevant literature [1–5,8,10], we identified the following variables as potential predictors of death: sex; age; physical nature, type, quantity, and concentration of caustic; type of ingestion (intentional or unintentional); systolic blood pressure ≤ 100 mmHg, (henceforth referred to as hypotension); heart rate ≥ 100 beats per minute in adults and over the expected value for age in children (henceforth referred as tachycardia); white blood cell (WBC) count; hemoglobin concentration; acidosis; intravascular disseminated coagulation; oropharyngeal burns; concomitant diseases; chest radiographs suggestive of aspiration or pneumonia.

The amount and concentration of caustic agents and the presence of oropharyngeal burns were inconsistently reported and were therefore not considered. Arterial blood gas measurements, coagulation tests and a standard chest radiograph had been obtained only in selected patients, so we did not consider them further. We assumed as major comorbidity the presence of any potentially life-threatening diseases in need of immediate therapy, including insulin-dependent diabetes mellitus. Only values of systolic blood pressure, heart rate, hemoglobin concentration and WBC count obtained immediately on admission were evaluated as predictors of death.

EGDs were carried out by eight operators trained by the same endoscopist (G.P.R.). Operators refrained from forcing the scope through the pylorus in cases of severe gastric damage. On the basis of agreement reached by the endoscopists of our unit [10], EGD reports gave information about mucosal injury and esophageal and gastric peristalsis. Mucosal injury was graded as follows, according to the method of Zargar et al., with minor modifications [9,13,14]: grade 1 (Zargar's grade 2a), friability, exudates, erosions, white membranes and superficial ulcers; grade 2 (Zargar's grade 2b), deep or circumferential ulcers; grade 3 (Zargar's grade 3a and 3b), areas of brown-black or grayish discoloration, taken as evidence of necrosis. For the early cases, occurring before Zargar's publication of 1989, the grade of mucosal injury was retrospectively attributed, based upon the original endoscopic reports. Esophageal, gastric and duodenal injuries were graded separately. The absence of peristalsis was diagnosed after 30 s of observation during gentle and careful insufflation by the endoscope. Esophageal and gastric peristalsis were considered separately.

Statistical Analysis

Comparisons of continuous variables (age, WBC count, hemoglobin level) between survivors and nonsurvivors were done using the Mann-Whitney *U* test, since these variables were not normally distributed. The chi-squared test (with Yates' correction when required) or the Fisher's exact test were used to compare the frequency of death in patients with and without the potential risk factor. A value of $P < 0.05$ was taken as statistically significant. The Mantel-Haenszel procedure was employed to control for the confounding effect of endoscopic variables on the risk of death in patients stratified for the degree of gastric injury. Predictors of death were then entered in a multivariate logistic model employing a stepwise procedure for selection of variables. A variable was included at each step if the score statistic was < 0.1 and removed if the log likelihood ratio statistic was > 0.2. The goodness of fit of the final model was tested using the Hosmer-Lemeshow test [15].

A risk score system was devised using the variables which had been entered into the final multivariate model. We assigned a score to each category of the predictor on the basis of its relative contribution to the logistic model, as determined by its regression coefficient. Score values were rounded to ± 0.5 units. The receiver operating characteristic (ROC) curve for the risk of death was drawn, and the area under the curve (AUC) was taken to be a measure of the ability of the score system to distinguish survivors from nonsurvivors.

Statistical analysis was done using the PEPI software package (USD Inc., West Park Place, Stone Mountain, Georgia, USA) [16].

Results

Patient Characteristics and Outcome

During the study period, 210 patients were admitted to our hospital within 12 h of caustic ingestion and underwent EGD. The endoscope was not forced through the pylorus in 34 patients (16.2%). No complications occurred as a consequence of EGD. In total, 13 patients underwent emergency surgery (6.2%). Table **1** shows details of patients who underwent surgery, or died.

The most seriously injured patients were fed by a surgically created enteral route or by total parenteral nutrition for at least 15 days and returned to nutrition by mouth only after endoscopic evidence of healing. Medical management included intravenous H_2 antagonists and in the most seriously injured patients, antibiotics and antifungal agents. A total of 25 patients (11.9%) died during their hospital stay. Although the timespan of the study was long, we did not detect a significant change in mortality during the 17-year study period (Mantel's chi-squared for the trend, $P = 0.091$).

Selection of Predictors

Eight out of 86 male patients and 17 out of 124 female patients died ($P = 0.45$). The median age of the patients was 42 years (range 1–88). A total of 34 patients (16.2%) were less than 18 years old and all of these survived. Survivors had a median age of 37 years (range 1–88) compared with a value of 65 years (range 21–78) for nonsurvivors ($P < 0.001$, Mann-Whitney *U* test). The linearity of the age-logit (logit, log[p/1-p]) of death

Table 1 Details of patients who underwent surgery or died

Initials	*Age*	*Grade of injury*	*Local complication*	*Surgery*	*Day of surgery*	*Hospital stay, days*	*Cause of death*
M.A.	21	E2; S3; D0	B + P	Gastrectomy + anastomosis	16	69	MOF
E.M.	63	E2; S3; D1	B + P	Gastrectomy + splenectomy + anastomosis	11	20	MOF
M.G.	74	E2; S3; Dne	B	Gastrectomy + anastomosis	1	3	MOF
R.B.	50	E2; S3; D3	P	Gastrectomy + splenectomy + abdominal esophagectomy	2	9	MOF
D.G.	64	E2; S3; D1	B	Gastrectomy + abdominal esophagectomy	1	22	MOF
P.M.	42	E1, S3; Dne		Gastrectomy + anastomosis	1	62	RI
O.B.	55	E2; S2; D0		Gastrectomy + anastomosis	1	11	MOF
R.I.	52	E3; S2; Dne		Gastrectomy + abdominal esophagectomy	1	3	MOF
P.R.	65	E1; S3; D3		Gastrectomy + abdominal esophagostomy	1	1	MOF
N.B.	6	E3; S2; Dne	B	Gastrectomy + esophagectomy with thoracotomy	1	77	Alive
E.F.	54	E2; S2; D1		Explorative laparotomy	1	89	Alive
A.S.	34	E2; S3; D0		Gastrectomy + abdominal esophagectomy	1	21	Alive
O.M.	48	E2; S3; D0		Cholecystectomy for acute cholecystitis*	25	68	Alive
C.B.	48	E3; S2; D0	B	No		13	B
O.B	75	E1; S1; D0	B	No		14	B
I.T	71	E3; S3; Dne	B	No		1	B
A.B.	62	E1; S3; D1		No		2	RI
D.C.	73	E1; S2; D0		No		10	RI
M.D.	75	E2; S3; D0		No		34	RI
G.G.	59	E2; S3; D1		No		15	MOF
R.G.	75	E2; S3; D2		No		5	MOF
C.L.	76	E2; S2; Dne		No		2	HF
R.M	66	E1; S1; D0		No		1	RI
G.N.	66	E2; S1; Dne		No		8	MOF
M.R.	78	E1; S1; D0		No		6	RI
A.S	59	E1; S3; Dne		No		5	MOF
E.T.	77	E0; S1; D0		No		4	RI
B.V.	53	E3; S3; Dne		No		2	MOF
R.V.	65	E2; S3; D0		No		3	MOF

Grade of injury: E, esophagus; S, stomach; D, duodenum. The number represents the grade of injury (for details of grading see Patients and methods section); ne, not explored. Local complications: B, gastrointestinal bleeding; P, perforation (not diagnosed by plain abdominal films on admission). Strictures are not reported in the table. Surgery: Gastrectomy + anastomosis is esophagus-jejunal anastomosis. No total esophagectomies were performed. Late surgery for treatment of strictures is not dealt with in the table. Day of surgery: the number of days from ingestion to when surgery took place. Cause of death: MOF, multiple organ failure; RI, respiratory insufficiency, including acute respiratory distress syndrome (ARDS) and pneumonia; HF, acute heart failure; B, gastrointestinal bleeding.
* The patient had been fed by total parenteral nutrition for 20 days.

relationship was tested using the Box-Tidwell procedure [15]. Age was treated as linear in logistic regression analysis and subdivided into 10-year intervals.

The type of ingested caustic is reported in Table **2**. All the ingested substances were liquids. Out of the 59 patients who had ingested strong acids, 20 died (33.9%), as did four out of 140 (2.9%) who had ingested other caustics. The relative risk (RR) of death associated with the ingestion of strong acids was 12.2 (95% CI, 10.6 to 13.8). Intentional ingestion was acknowledged by the patient or witnessed by friends or relatives in 127 cases (60.5%). Out of the 25 deaths, 24 were due to intentional ingestion (RR = 15.7; 95% CI, 13.6 to 17.7). No death was observed among the 10 patients who accidentally ingested a strong acid, while 20/49 patients (40.8%) who deliberately ingested such a caustic died (P = 0.012, Fisher's exact test). Regarding the ingestion of caustics other than strong acids, 1/67 patients with accidental ingestion died, compared with 3/73 patients where ingestion was deliberate (P = 0.621, Fisher's exact test).

Table 2 Types of caustics ingested

Caustic type	*Patients*		*Nonsurvivors*	
	n	*% of total patients*	n	*% of those ingesting specific caustic*
Strong acids	59	28.1	20	33.9*†
Strong alkalis	36	17.1	2	5.6
Ammonia	14	6.7	1	7.1
Bleach	65	31.0	1	1.5
Others	25	11.9	0	0.0
Unknown	11	5.2	1	9.1
All types	210	100	25	11.9

* $P < 0.0001$, death rate of patients who ingested strong acids vs. those who ingested other caustics; † $P < 0.01$, death rates of patients who ingested strong acids vs. those who ingested strong alkalis.

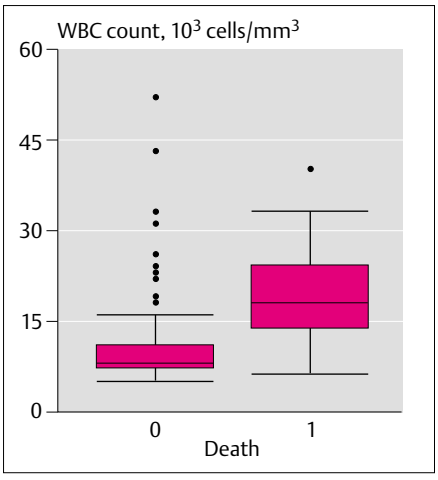

Figure 1 White blood cell (WBC) count distribution in survivors (death = 0) and nonsurvivors (death = 1). WBC are expressed as × 1000 cells/mm³. The length of the box represents the interquartile range (IQR), which contains the middle 50% of the data (from the 25th to the 75th percentile). The line through the inside of the box represents the median value. The adjacent values are represented by the T-shaped lines that extend from each end of the box, from the largest observation which is less than or equal to the 75th percentile plus 1.5 times the IQR, to the smallest observation which is greater than or equal to the 25th percentile minus 1.5 times the IQR. Values outside this range, "outliers", are represented as individual points

The relationship between outcome and WBC count at admission is shown in Figure **1**. The median WBC count on admission was 8850 cells/mm³ (range 4500 – 52 000). Survivors had a median WBC count of 8400 (range 4500 – 52 000), compared with a value of 17 050 cells/mm³ (range 5800 – 40 000) in nonsurvivors ($P < 0.0001$, Mann-Whitney *U* test). Since WBC count was not linearly related to the logit of death, we chose a value of 20 000 cells/mm³ as the cutoff point for our models: 10 out of 21 patients with WBC count ≥ 20 000 cells/mm³ died, and 15 out of 189 with WBC count < 20 000 cells/mm³ (RR = 6.0; 95% CI, 5.3 to 6.7).

Hemoglobin values were not significantly different in survivors (median 13.0 g/dL) and nonsurvivors (median 13.5; $P = 0.82$, Mann-Whitney *U* test).

Six patients were affected by ischemic heart disease or cardiac insufficiency, four by chronic obstructive pulmonary disease, two by insulin-dependent diabetes, and two by alcoholic liver disease. One patient had a fall as a sequel of caustic ingestion and developed an epidural hematoma. Another patient took a large dose of barbiturates. Six out of 14 patients with major comorbidities died (two patients had two comorbidities each), and 19/196 without comorbidities (RR = 4.4; 95% CI, 3.9 to 4.9). Four patients were hypotensive at admission and all survived. Five out of 14 patients with tachycardia and 20/196 with normal cardiac rate died (RR = 3.5; 95% CI, 3.1 to 3.9).

Table 3 Endoscopic findings and patient outcomes

Endoscopic findings	*Patients, n*	*Nonsurvivors, n*
Location and type of lesion		
Esophagus		
No injury	102	1
Grade 1	68	7
Grade 2	27	12
Grade 3	13	5*
Stomach		
No injury	107	0
Grade 1	66	5
Grade 2	9	4
Grade 3	28	16*
Duodenum		
No injury	151	9
Grade 1	21	4
Grade 2	0	0
Grade 3	4	3†
Not explored	34	9‡
Absence of esophageal peristalsis	22	13§
Absence of gastric peristalsis	23	14§

* $P < 0.00001$, for comparison of death rates associated with different degrees of injury; † $P < 0.001$, for comparison of death rates associated with different degrees of injury; ‡ $P < 0.01$, for comparison of death rates in patients with explored vs. unexplored duodenum; § $P < 0.00001$, for comparison of death rates in patients with vs. without peristalsis.

In 79 patients (37.6%) there was no pathological finding or only diffuse hyperemia at EGD; none of them died. Table **3** shows the outcomes in patients classified by endoscopic findings. The degree of gastric injury was significantly associated with mortality ($P < 0.00001$). In the univariate analysis, esophageal injury, duodenal injury, absence of esophageal peristalsis, absence of gastric peristalsis, and lack of duodenal exploration, were also associated with death. However, these variables were not confirmed as independent predictors of death when data were stratified according to degree of gastric injury. The caustic type had no influence on the location of lesions (data not shown).

Nine out of 13 (69.2%) surgically treated patients died compared with 16/197 (8.1%) patients managed conservatively (RR = 8.52; 95% CI, 7.4 to 9.6).

Predictors of death found by univariate analysis were entered in a stepwise logistic regression model (Table **4**). Surgery was not identified as a significant predictor of death by logistic modelling; this was still the case after exclusion of the patient who underwent cholecystectomy (not shown). We did not include the variable "type of ingestion" (intentional/unintentional) in the model, because it was highly co-linear with the ingestion of strong acids. For the logistic regression analysis, gastric injury was coded as follows: no injury or grade 1 (reference group); grade 2 (deep ulcers); and grade 3 (necrosis). Patients with ingestion of unknown caustics were excluded from the analysis. This exclusion left 199 patients and 24 deaths available for analysis. Age, ingestion of strong acids, WBC count ≥ 20.000 cells/mm³ and grade of gastric injury were identified as independent predictors of death by the final model. The Hosmer–Lemeshow test confirmed the goodness of fit of the final model (p is near to 1).

Table 4 Logistic regression model for the estimation of death risk

	β	*SE*	*P value (Wald test)*	*OR*	*95% CI*
Variable					
Age, 10-year interval*	0.86	0.28	0.002	2.37	1.37 to 4.10
Strong acid ingestion	2.07	0.76	0.007	7.93	1.78 to 35.33
WBC count ≥ 20 000/mm^3	1.79	0.78	0.02	6.02	1.29 to 28.02
Gastric deep ulcers†	2.28	0.98	0.02	9.7	1.42 to 66.85
Gastric necrosis†	3.03	0.75	0.0001	20.88	4.75 to 91.83
Constant	– 9.59	2.02	0.00001		

β, logistic regression coefficient; SE, standard error of β; OR, odds ratios; 95% CI, 95% confidence interval of OR. WBC, white blood cell. *Continuous variable. †When both ulcers and necrosis were present, only necrosis (the most severe lesion) was considered.

Table 5 The risk scoring system

Variable	*Score*
Age (for each 10-year interval)	1
Type of caustic	
Other than strong acids	0
Strong acid	2
WBC count at admission	
< 20 000/mm^3	0
≥ 20 000/mm^3	2
Gastric injury*	
No injury or grade 1	0
Grade 2 (deep ulcers)	2.5
Grade 3 (necrosis)	3

*Grade 1 corresponds to Zargar' s grade 2a; grade 2 to Zargar's grade 2b; grade 3 to Zargar's grades 3a and 3b (see text for details).

Table 6 Number of deaths for different scores

Score	*Patients*	*Nonsurvivors*		*Positive likelihood ratio**
		n	%	
<10	166	0		0
10 – 11.9	13	7	53.8	8.5
12 – 13.9	9	7	77.8	25.5
≥14	11	10	90.9	72.9
All patients	199	24	12.1	

*Positive likelihood ratio for risk of death. Pre-test probability 12.1%.

Risk Score System

The score system devised from the results of the logistic regression analysis is shown in Table **5**. Table **6** shows the mortality for each score category. No patient scoring less than 10 points died, and just one of the patients scoring 14 points or more survived. The ROC curve was drawn (not shown), and the AUC, taken as a measure of the discriminatory ability of the score system for predicting the risk of death, was 0.99 (95% CI, 0.979 to 1; standard error 0.006), corresponding to a satisfactory discriminatory ability.

Discussion

Our study confirms the reproducibility of Zargar's endoscopic classification [9,13,14] of caustic burns and its ability to predict the risk of death. Furthermore, our results show that age, ingestion of a strong acid, WBC count ≥ 20 000 cells/mm^3, and the presence of gastric deep ulcers or gastric necrosis at EGD are independent predictors of death after caustic ingestion. A scoring system based on these variables accurately distinguished survivors from nonsurvivors. The patient's age, WBC count, and type of ingested caustic can easily be obtained from history and laboratory exams; in addition, we believe that even an endoscopist not specifically trained to do so can recognize the deep gastric ulcers and areas of gray or brown-black discoloration which are the hallmark of severe gastric injury. The simplicity with which the variables of our score system can be obtained may promote its use in the emergency department setting for the triage of patients after caustic ingestion.

The prognostic relevance of leucocytosis was suggested in previous reports [5]. In our study, blood samples were taken on admission, which occurred within 12 h from ingestion; thus we suggest that a WBC count ≥ 20 000 cells/mm^3 in any blood specimen taken within 12 h of caustic ingestion should be considered a predictor of a poor outcome.

According to our experience, young patients with ingestion of weak acids or alkalis and with low WBC counts have a low risk of death, and one may wonder whether these patients need to undergo EGD. Out of 1730 cases of fatal intoxication due to corrosive aromatic substances, caustic acids or alkalis reported between January 1982 and December 1994 in Italy, only 40 (2.3%) involved patients less than 18 years old [17]. These data suggest that although caustic ingestion is a frequent household accident in childhood [18] and may cause esophageal or gastric strictures, it is rarely responsible for death. Authors disagree about whether all children with suspected caustic ingestion should undergo EGD [2,4,19,20], and further studies are needed.

It is not surprising that all the instances of fatal strong acid ingestion were intentional: the pungent smell and taste of these agents usually prevents accidental bulky ingestion [18].

Among the endoscopic variables, only severe gastric injury was entered into the final prediction model. In our patients, esophageal injury was often milder than gastric injury and duodenal necrosis was detected only in four cases. It should be noted that all the caustics were liquids and that selective esophageal involve-

ment with sparing of the stomach has been reported in particular for lye in the solid state [21].

Our study has some limitations, related in part to its retrospective design. We were not able to evaluate all the potential clinical predictors identified by other authors. Only a few patients were reported to be affected by life-threatening comorbidities. However, collecting an accurate medical history in the emergency department may be difficult, especially with patients who are experiencing severe pain and psychological distress. It is thus possible that the contribution of age may partly arise from the presence of associated comorbidities.

To our knowledge, an investigation of the agreement among different endoscopists concerning the assessment of caustic lesions has never been performed. The operators in our study had all been trained by the same physician and they described upper gastrointestinal tract findings according to a protocol, which has been adopted by our unit since its earliest days. We did not consider mucosal hyperemia or edema, which are characteristic of the first grade in Zargar's original classification [9], because of the high interoperator variability associated with the diagnosis of these lesions. In addition, extensive and scattered necrosis were considered together because they may be difficult to distinguish, especially when the presence of clots and food does not allow a complete exploration. Assessment of peristalsis by endoscopy is not fully accepted, but we and others [6,10,11,22] have proposed that the absence of peristalsis is characteristic of the most severe cases of caustic burns.

The duodenum was not explored in all our patients. If the stomach is severely damaged, entering the duodenum with the endoscope may be dangerous. Relative sparing of duodenum, as compared to esophagus and stomach, has been reported as a consequence of protection offered by pyloric spasm [13,18]. It is not known however, whether forcing the pylorus some hours after caustic ingestion might increase dispersion of the caustic material.

We did not find significant differences in mortality over the long study period. Nutritional supplementation is considered to have been a major improvement in the management of patients with caustic burns [7], but it had already been adopted at our institution before the beginning of the present study.

Our study was not designed to identify the patients most likely to benefit from early surgical treatment. Before a decision is made for surgery, an accurate assessment of the severity of the injury is needed, including standard chest radiography, EGD, laryngoscopy, and tracheobronchial endoscopy in selected cases [5]. The presence of esophageal and duodenal necrosis and lack of esophageal and gastric peristalsis were not independent predictors of death in our model, even though they have been reported as being of value in making the decision for surgical treatment [5,10–12,23].

The long timespan needed to collect a sufficient number of patients hampered us in prospectively validating our risk score system in a further series of patients. At multivariate analysis, surgery was not identified as an independent predictor of death in our patients. It should be noted that the majority of our patients were managed conservatively. Only six patients underwent surgery upon the basis of endoscopic findings before the diagnosis of perforation, gastrointestinal bleeding, or acute cholecystitis (see Table **1**). This may partly explain the poor outcome in surgically treated patients. Early aggressive surgical treatment by a fully trained surgical team has been reported to be life-saving in selected cases of severe caustic injury [5,11,23–25], but few prospective studies have been conducted so far. We do not know whether a more aggressive treatment could have been life-saving in some of our cases; if this is the case, the performance of our score system might be poorer in centers where there is a more aggressive use of surgery.

In summary, our study suggests that a simple scoring system based on clinical and endoscopic parameters can be used to predict the risk of death after caustic ingestion. This score system has a high internal validity but its usefulness for the triage and treatment of patients with caustic injuries should be prospectively confirmed on external samples at other institutions.

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