

Effectiveness of bougie dilation for the management of corrosive esophageal strictures

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Abstract

Background and study aims : Caustic ingestion caused by swallowing a detergent can produce a progressive and devastating injury in the esophagus and stomach. One of the most important outcomes of the corrosive oesophagitis is the stricture formation, which is resistant to treatment. The aim of this study was firstly to determine the relation between agent, inflammation and stricture, and secondly investigate the efficiency of dilation in patients having esophageal stricture due to corrosive oesophagitis.

Patients and methods : In this study, 58 cases with post caustic oesophagitis, which had been admitted to our clinic or emergency department between January 1999 and December 2004, were assessed retrospectively. Dilation of esophageal stricture of the cases was performed by Savary-Gilliard bougies.

Results : The most frequently ingested substance was alkaline (48.2%). Concerning all the patients, the most frequent location of caustic injury was upper esophagus (36.2%), and grade I injury was the most frequently encountered one (34.4%). Thirty patients (51.7%) developing stricture were treated by repeated dilations. The most common location of stricture was middle esophagus (50%), and severe stricture was the most common one among all stricture grades (46.7%). Alkaline ingestions yielded more severe stricture than acids. Eight of the patients with stricture (26.6%, 8/30), who didn't respond to periodic esophageal dilation, underwent esophageal resection or bypass surgery.

Conclusion : Dilation with Savary-Gilliard bougies is a quite effective method for stricture after corrosive oesophagitis. (*Acta gastroenterol. belg.*, 2006, 69, 372-376).

Key words : corrosive esophageal stricture, bougie dilation.

Introduction

Caustic ingestion caused by swallowing a detergent can produce a progressive and devastating injury in the esophagus and stomach (1). Caustic ingestions are generally associated with suicidal intent in adolescents and accidental in children (1,2). The severity of injury to the gastrointestinal tract depends on the ingested substance ; its amount, concentration and physical form (solid or liquid) and the duration of exposure (1). Superficial mucosal (first degree) injury, transmural mucosal injury with possible muscularis involvement (second degree) injury, or full thickness (third degree) injury may result. Alkalis may cause a deep liquefaction necrosis along intense inflammation and saponification of the mucous membranes, submucosa and muscularis, while acids typically lead to a more superficial coagulation necrosis with scar formation. Because there is no protective scar formation, the damage of gastrointestinal wall caused by alkali agents is frequently more severe than that of caused by an acid (1,3).

The pathologic classification of caustic injury to the esophagus is similar to classification of burns to the skin. This grading system for esophageal injury provides the prediction of subsequent clinical outcomes. Grade I injury looks like a first-degree burn. Grade II injury looks like a second-degree burn. Grade II burns were labeled by Zargar *et al.* grade IIA if patchy or linear, or grade IIB if the injury is circumferential (4). Grade III injury corresponds to a transmural burn and necrosis without perforation. Grade IV (perforation) is usually not encountered because radiographic and clinical findings are diagnostic and in the presence of high clinical suspicion to perforation oesophagogastroduodenoscopy is contraindicated (1).

The clinical features of caustic ingestions are varying widely. Caustic ingestion can result in acute (oesophagitis, gastritis, perforation, internal bleeding, sepsis) and chronic complications (oesophageal stricture, antral stenosis, oesophageal cancer and carcinoma of the stomach) (1). If the patient survives the acute effects, oesophageal and/or gastric stenosis can occur. Up to one-third of patients who suffer caustic esophageal injury develop esophageal strictures ; particularly those with grade IIB or III injury (1). Management of these conditions is very difficult. Treatments of esophageal strictures are dilation using a balloon and bougie or surgery (1,5). The literature on the treatment of these patients is quite controversial and inconclusive. There have been few studies with extensive series that have successful results (6-8). For this reason, we firstly aimed to determine relation between agent, inflammation and stricture, and secondly investigate the efficiency of Savary-Gilliard bougie dilation in patients with esophageal stricture due to corrosive oesophagitis.

Patients / material and methods

In this study, 58 cases with post-caustic oesophagitis, which had been admitted to our clinic or emergency department between January 1999 and December 2004,

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were assessed retrospectively from their medical records. Only, adults constituted our study group. Parameters included sex, age, caustic agent, the reason of ingestion, degree and site of burn and stricture, number of dilation, number of dilators being used were evaluated. All the patients were observed in endoscopic examination after swallowing corrosive substances.

Oesophagus was divided into three parts. Upper (part 1), middle (part 2) and lower (part 3) oesophagus were 1-17 cm, 17-24 cm, and 24-40 cm, respectively. Injuries were classified according to the method of Zargar (4). Strictures were classified following the bougie size used in the course of the dilation as mild (11-13 mm), moderate (7-11 mm) and severe (< 7 mm).

Dilation with Savary-Gilliard bougies was performed twice a week to the patient who developed stricture during the follow-up period. Our end-point on dilation was to achieve an esophageal lumen of 15 mm in diameter or make the patient asymptomatic. Patients were then followed with monthly controls. The numbers of dilation sessions were grouped as follows ; 0-5 sessions, 6-10 sessions, 11-15 sessions, and more than 15 sessions, respectively. Before and after dilation, anti-reflux treatment with gastric acid secretion inhibitors or sucralfate were given to the patients. Patients who didn't respond to dilation underwent surgery.

Statistical analysis

All statistical analyses were performed by SPSS 11.5 (SPSS Inc. Chicago, IL., USA) software. Descriptive data were quoted as either the mean \pm SD or median (range) notation. Frequencies and percentages were calculated. Relations among the categorical variables were investigated with Chi-Square test. P value was set at 0.05.

Results

A total of 58 patients were evaluated : 47 patients (81.1%) were men and 11 patients (18.9%) were women. Ages ranged from 20 to 63 (median = 23). Ingestion was associated with accidental in 55.2% of cases (32/58) and suicidal intent in 44.8% of cases (26/58). Suicide attempt was frequent in women (63.6%, 7/11), and accidental corrosive ingestion was frequently

observed in men (55.3%, 26/47). The most ingested agent was alkali (48.2%, 28/58) and rate of acids was 39.6% (23/58). The kind of remaining agents was unknown (12.2%, 7/58).

The most frequently encountered mucosal injury was grade I (34.5%, 20/58). Grade IIA, grade IIB and grade III injuries were documented in 15, 12, and 10 patients (25.9%, 20.7%, 17.2%), respectively. One patient (1.7%) with oesophagus perforation determined by endoscope was immediately operated on. Concerning all the patients, injuries were in more than one third of the oesophagus, with 36.2% (21/58) frequency of lesions involving the upper third, 27.6% (16/58) involving the middle third and 8.6% (5/58) involving the lower third. In 27.6% of cases (16/58), the entire of oesophagus was involved. Stomach was involved in %15.5 (9/58) of cases. All of them had mild mucosal injuries and the most ingested agent was acid (55.5%, 5/9).

Stricture developed in 51.7% of cases (30/58). Stricture was mild in 23.3% of cases, moderate in 30%, and severe in 46.7%. The incidence of stricture was 27.2% in women and 57.4% in men (Table 1). The most site of stricture was the middle third of the oesophagus (50%, 15/30). Distribution of the location of stricture according to caustic substance was shown in Table 2.

Over the 5 years review, no specific treatment was necessary for the 20 patients with grade I injury and the 7 patients in grade IIA injury. Mild stricture formation was observed in 7 patients with grade IIA injury. Moderate stricture formation was seen in 1 patient with grade IIA injury and in 8 patients with grade IIB injury. Four patients with grade IIB injury and all the patients with grade III injury had severe stricture.

Patients developing stricture were treated by repeated dilations. The median number of dilation sessions was 6 (1-36). Although the frequency of dilation would decrease over the first 6 months, two patients (6.6%, 2/30) continued to require dilation procedures over 5 years after their initial injury. Complications did not develop in endoscopic intervention cases. 26.6% of the patients (8/30) with stricture, who didn't respond to periodic oesophageal dilation, underwent oesophageal resection or bypass surgery.

Alkali ingestions yielded more inflammation in middle oesophagus (50%), acid in upper esophagus (46.7%) ($\chi^2 = 19.400$, $p = 0.15$). Oesophageal stricture was severe

Table 1. — Frequency distribution of stricture in female and male patients

Sex	Female		Male		Total	
	n	%	n	%	n	%
Stricture						
Mild			7	26.0	7	23.3
Moderate			9	33.3	9	30.0
Severe	3	100.0	11	40.7	14	46.7
Total	3	100.0	27	100.0	30	100.0

Table 2. — Distribution of the location of stricture according to caustic substance

Caustic agent	The location of stricture in the esophagus													
	Part 1		Part 2		Part 3		Part 1 and 2		Part 1 and 3		Part 2 and 3		Total	
Stricture	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Alkali	6	75.0	8	53.3	2	66.7	1	50.0	1	100.0	1	100.0	19	63.4
Acid	2	25.0	4	26.7	1	33.3	1	50.0					8	26.6
Unknown			3	20.0									3	10.0
Total	8	100.0	15	100.0	3	100.0	2	100.0	1	100.0	1	100.0	30	100.0

Table 3. — Distribution of the severity of stricture according to caustic substance

Caustic substance	The severity of stricture							
	Mild		Moderate		Severe		Total	
	n	%	n	%	n	%	n	%
Alkali	4	50.0	5	62.5	10	71.4	19	63.4
Acid	3	37.5	1	12.5	4	28.6	8	26.6
Unknown	1	12.5	2	25.0			3	10.0
Total	8	100.0	8	100.0	14	100.0	30	100.0

Table 4. — Distribution of dilation according to caustic substance

Caustic substance	Number of dilation									
	0-5		6-10		11-15		> 15		Total	
	No	%	No	%	No	%	No	%	No	%
Alkali	6	60	2	66.6	6	85.7			14	63.6
Acid	3	30	1	33.3	1	14.3	2	100.0	7	31.8
Unknown	1	10							1	4.6
Total	10	100.0	3	100.0	7	100.0	2	100.0	22	100.0

in all women and 40.7% of men (11/27). Sex was not correlated with the severity of stricture ($\chi^2 = 3.810$, $p = 0.149$). Alkali ingestions caused more severe stricture than acids ($\chi^2 = 2.381$, $p = 0.666$) (Table 3).

Sex was not correlated with the type of corrosive agent, number of dilation session, and number of dilators ($p > 0.05$). Corrosive substances were not correlated with the location and severity of stricture, number of dilation session (Table 4 contains the patients who were cured), and number of dilators ($p > 0.05$).

Discussion

The objective of this study was to analyze a 5-year historical series of patients treated in our clinic who ingested caustic substances, and to assess the effectiveness of dilating therapy with Savary-Gilliard bougies administered in patients with strictures after caustic injury in oesophagus.

Our study population had two distinctive characteristics: a male/female ratio of 4.3 and a young median age. It is well known that caustic ingestion is more frequent in children. Campbell *et al.* reported that 80% of hospitalized patients with corrosive oesophagitis were less than 10 years age (10).

A strong alkaline or acidic agent produces caustic injury. Alkali solutions are often odorless and tasteless and are swallowed before protective reflexes can be evoked. Acidic agents usually cause immediate pain and if not intentional, the agents are rapidly expelled (1). It is known that alkaline are more frequently swallowed than acids. Bozymisky *et al.* stated that acid ingestion occurred in approximately 15% of caustic injuries (11). Acid ingestion rate was higher in our study, maybe linked to higher rates of suicide attempts.

Patients who ingest caustic agent may appear in any condition from asymptomatic to frankly toxic with perforation findings. After resolution of the initial injury, oesophageal stricture and oesophageal squamous carcinoma may emerge. In the literature, the ratio of clinically apparent oesophageal stricture shows some discrepancies: Yong *et al.* (7) and Gundogdu *et al.* (12) have found high incidence rates of oesophageal stricture (85% and 72.7%, respectively). Symbas and coworkers reported low esophageal stricture rates in 39.2% of their patients (13). In our study, stricture developed in 51.7% of the patients (30/58). Analysis of the patient's group that developed stricture showed that the middle third was involved on 50% of the cases, followed by the upper third in 26.6%, the lower third in 10% and by two parts

in 13.3%. Gundogdu's group indicated that children have stricture of the upper third instead of the middle third lesions shown in the present study (12).

The stomach was injured in 15.5% of the cases in our group and it did not result in antral stricture during the follow-up. Some investigators stated that antral stricture might develop rapidly 3 to 6 weeks after the injury but in some cases it might appear after several years (1,14). Duodenum was not involved in contrast to other studies (15). Perforation can occur in any time within the first two weeks following the injury and during dilating treatment (1). One (1.7%) patient with perforation based on first endoscopic examination underwent surgery.

The initial treatment of caustic ingestion is observation, with precaution of vomiting, choking, and aspiration. Emergency surgery is indicated in cases of oesophageal and gastric perforation (1). Medical treatment of caustic ingestion includes corticosteroids, antibiotics, H₂ antagonists, and sucralfate. The role of steroids is controversial. Although some authors report that glucocorticoids may reduce the incidence of strictures (2,16), Oake's review of the literature reported that the use of steroids was not beneficial (17). Antibiotics should be used if specific indications develop. However, some investigators recommend the systematic use of prophylactic antibiotics (18,19). Gastrooesophageal reflux has a tendency to worsen the caustic injury to the esophagus. It may accelerate stricture formation. Using of H₂ receptor blockers, or proton pump inhibitors, and sucralfate may be a good way to maintain good acid control in patients with caustic ingestion (20,21).

Dysphagia symptom gives a signal for the onset of an esophageal stricture and develops in 15% to 38% of cases with caustic exposure (1). Dysphagia typically manifests between 4 and 6 weeks with the development of obstruction from scar formation after the initial injury but stricture may not become apparent for a year (22). The primary treatment for caustic esophageal strictures is dilation with bougie or balloon. In our study all patients showed immediate improvement: 66.6% (20/30) were cured by repeated dilations and 6.7% (2/30) continued to require dilation therapy over 5 years after injury, but 8 patients (26.7%, 6/8 alkaline, 2/8 acid, 1/8 unknown) required surgical intervention. We did not encounter major complications during dilation session in cases, i.e. perforation.

Although endoscopic balloon dilation has been recently described, bougies are generally used for dilating oesophageal strictures (23,24,27). It has been suggested that the radial force applied by distending a balloon is more effective and safer than the abrupt shearing axial force of a bougie, but these claims have not yet been proven (28). Balloon dilation is not the treatment of choice in our setting. Firstly, it is costly and it is recommended that the balloon be used only once; secondly, the literature shows no significant difference between bougie and balloon (23,27). Furthermore, corrosive strictures tend to be extensive, multi-segmented, rigid,

tortuous, and technically difficult to dilate (29-31). Because of these reasons, a bougie were felt essential when tight and complex strictures are being treated.

All the patients with strictures due to an alkaline agent responded to bougie dilation treatment and were cured. On the contrary the therapy had to be continued in two patients who ingested acid (Table 4). Interestingly, one of these patients was a 32-year-old pregnant woman with an oesophageal stricture caused by accidental ingestion of Fehling's solution. Bougie dilations were applied to this patient both pre and post-delivery periods. Monthly controls are going on for the case.

Other endoscopic treatments for esophageal stricture have been recently described in the literature. Self-expanding and removable plastic stents can be used, reducing the number of dilation sessions with longer patency periods of the oesophageal lumen, and without the usual complications seen in malignant strictures (32,33). Early dilation starting immediately after injury results a high incidence of perforation, and is not currently recommended (34). The intralesional use of corticosteroids (triamcinolone) in complex esophageal strictures has attracted attention. However, the results are still controversial and the effectiveness of triamcinolone was not clearly proven (31,35).

Some patients fail to respond to bougie dilation and require gastrostomy, or oesophagectomy and reconstruction with colonic or jejunal interposition grafts (36). Surgical intervention is a good way to solve long and severe strictures. Adegboye *et al.* reported that oesophageal replacement was carried out in more than half patients with established oesophageal strictures (37). Surgical treatment is a good option in patients with severe strictures (10).

Patients with oesophageal burns should be watched throughout their lifetime for the late complications such as oesophageal stricture, antral stricture, carcinoma of the esophagus and stomach. Treatment of strictures after oesophageal injury is difficult, with a preference given to bougie dilation in many centres. The optimal frequency and time length should be individualized. The time interval between dilations depends on the effect of previous dilation effect and complications. Dilation therapy with Savary-Gillard bougies seems to be an appropriate management to solve the problem of strictures in a majority of patients.

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