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ScienceDirect
Digestive and Liver Disease 41 (2009) 263–268
Digestive and Liver Disease
www.elsevier.com/locate/dld

Alimentary Tract

Corrosive oesophageal strictures in children: Outcomes after timely or delayed dilatation

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Received 15 May 2008; accepted 25 July 2008
Available online 17 September 2008

Abstract

Background and study aims. Among benign oesophageal lesions, caustic strictures are the most difficult to dilate. In low-income countries, children suffering caustic oesophageal injury are frequently referred to the hospitals late, sometimes weeks after ingestion. Therefore, dilatation may be performed late and in highly fibrotic strictures. Reports about endoscopic and clinical outcome of such delayed dilatations are scanty. The aim of this study was to evaluate the safety and efficacy of late caustic stricture dilatations in children, comparing it with the results of timely dilatations, both performed at the Hospital of the Italian Non-Governmental Organization "Emergency" at Goderich, Sierra Leone.

Patients and methods. From December 2005 to May 2007, 78 children (<15 years) complaining alkaline caustic ingestion were submitted to oesophageal dilatation, mainly (97%) using Savary dilators. Two groups were identified: children (group 1) with a late treatment (>6 weeks, 37 ± 12 weeks), having arrived to the hospital late after ingestion, and children (group 2) dilated timely, i.e. at <6 weeks (4 ± 1.4 weeks) after injury.

Results. Strictures were severe in all patients. Twenty-five children were dilated *late* after injury (6.4 dilatations/patient) with a follow-up of 11 ± 2.5 months. A successful clinical outcome was observed in 91.6%. Four perforations (2.6% procedure-related) and one death (4.0%) were observed. Strictures recurred once in 72% of patients, twice in 31.8%. Thirty-one children were dilated *timely* (4.5 dilatations/patient) with a follow-up of 10 ± 2.1 months and a clinical success rate of 96.7%. Procedure-related perforation rate was 0.7% with one death (3.2%). Stricture recurred once in 30% and twice in 3.3%.

Conclusions. Delayed dilatation of caustic oesophageal strictures in children carries a higher risk of perforation and a higher recurrence rate.

Keywords: Children; Dilatation; Oesophageal caustic lesions; Oesophageal strictures

1. Introduction

During the past decades the incidence of oesophageal corrosive injuries in children has been reduced, especially in Western countries [1]. As a consequence, the individual experience in managing the resulting strictures, considered to be most difficult to treat [2], is limited in developed countries, where the acute management, as well the possible dilatation treatment, are usually carried out in a timely manner. Conversely, in low-income countries, children suffering these injuries are treated not infrequently at home, sometimes by traditional healers, and are often referred to the hospital weeks or months after injury. Moreover, endoscopic and dilatation facilities are seldom available in district hospitals, thus further delaying a proper treatment. In such settings, oesophageal dilatations might be performed at a (very) late stage, when the stricture is already well established, highly fibrotic and frequently accompanied by a significant anatomical distortion of the oesophageal lumen. Little is known about

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1590-8658/$30
doi:10.1016/j.dld.2008.07.319

the clinical and endoscopic outcome of late oesophageal dilatations compared to those performed in a timely manner [3].

Due to the practice of making soap at home in the African country of Sierra Leone, corrosive injuries are particularly frequent because of the wide availability of this agent in open containers at home. Since endoscopic and dilatation facilities became available in our surgical centre in Goderich, Sierra Leone, in December 2005 it has become possible to manage children with old corrosive oesophageal injuries as well as new patients referred to the hospital shortly after ingestion. This gave us the opportunity to evaluate and compare the endoscopic and clinical outcome of two groups of patients: those who had timely dilatation and those treated late after injury.

Hence, we report an observational study dealing with two groups of patients with oesophageal strictures. In the first group the children arrived at the hospital after a long (>24 weeks) interval time since ingestion and had a late dilatation treatment, without any early diagnostic endoscopy; in the second group, the patients were timely dilated, according to the literature [4]. Several patients of this last group were submitted to a diagnostic endoscopy within 48 h after the ingestion. Because the goal of dilatation, in addition to the immediate relief of dysphagia, is to provide a long dysphagia-free interval, recurrence of dysphagia was taken as the end point for comparing the efficacy of dilatation.

2. Patients and methods

In December 2005 the Surgical Center of the Italian Non-Governmental Organization (NGO) "Emergency" at Goderich-Freetown, Sierra Leone was supplied with fibre-optic endoscopes (Olympus GIF-NX30) as well as with dilatation devices. Since then, all children (<15 years) presenting with recent (within 48 h) accidental caustic ingestion admitted to the hospital were submitted to an early endoscopy. When a trivial lesion or no lesions at all were found, the patients were discharged. If a second or third degree (according to Kirsh and Ritter [5]) oesophageal lesion was observed at endoscopy, a surgical gastrostomy was performed. Antibiotics (ampicillin + cloramphenicol), intravenous antisecretory compounds (H_2-receptor antagonists or proton pump inhibitors, when available) and intravenous fluids were given in the acute post-injury phase. Corticosteroids were administered only in case of upper respiratory symptoms. The first dilatation was scheduled at least 3 weeks after injury.

When the patients were admitted more than 3 days after ingestion, an endoscopic examination was not carried out, for the fear of perforation, and a surgical gastrostomy was performed when the children could not swallow liquids or saliva. Endoscopy and eventual dilatation was delayed for 3 weeks after injury. If the children were admitted at more than 3 weeks after ingestion, complaining dysphagia, an endoscopic examination and a dilatation attempt, if needed, were carried out as soon as possible. A gastrostomy was always performed in case of unsuccessful or very difficult dilatation and when the children were severely malnourished.

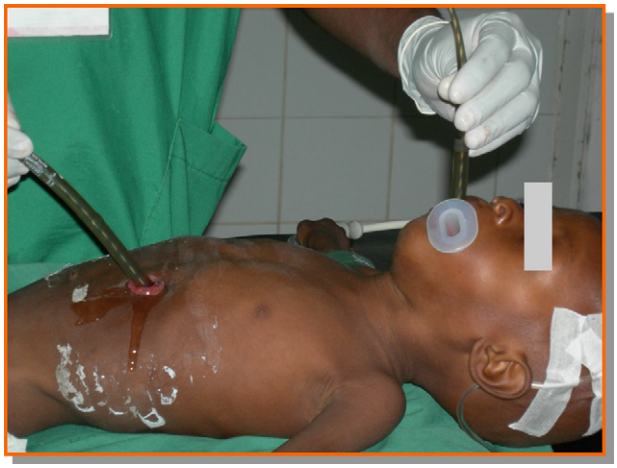

Fig. 1. Retrograde oesophageal dilatation *via* gastrostomy over a soft angiographic guide wire.

All children admitted to the hospital before December 2005 were recalled for clinical and endoscopic evaluation and subsequent dilatation, if needed. All dilatation procedures were carried out under general anaesthesia with intubation of the patients and Savary bougies were used when possible. The correct position of the guide wire across the stricture and into the stomach was confirmed by fluoroscopy or simply by palpation of the abdomen, as the tip of the guide wire abutted the anterior abdominal wall. If the stiff Savary guide wire was unable to traverse the stricture, a softer angiographic guide wire (0.21–0.25, 0.35 mm) was used. Softer guide wires, used only in the presence of a gastrostomy, came out from the gastrostromy itself and from the mouth (Fig. 1). They were kept strained by a physician assistant, thus reducing the risk of perforation. When it was impossible to overcome the stricture by a guide wire, a pneumatic dilatation was attempted. Oesophageal balloon (Rigiflex®) dilatation, performed only when the stricture was extremely narrow and tortuous, was mainly used as a mean of obtaining an initial access of the stricture. Dilatation was gradually extended downward under endoscopic surveillance. The passage of the guide wire and the dilatation were preferably carried out by a retrograde approach if a gastrostomy was present. Dilatations were performed every 1–3 weeks, keeping time flexible and based on the severity of symptoms and the degree of narrowing. Dilatation began with a 5 mm Savary bougie, increasing to not more than 4 mm in diameter each session. If frank blood stain was observed on the bougie's surface, dilatation was stopped. The treatment was considered fully adequate if the oesophageal lumen could be finally dilated to 11 or 12 mm. Balloon dilatation was stopped if frank blood or extended mucosal lacerations were observed through the transparent balloon membrane during

the procedure. Fluoroscopic control during balloon dilatation was not performed, because water-soluble contrast media to fill the balloons were not available. Intravenous antibiotics (ampicillin + cloramphenicol) were administered at 1 h before the procedure and continued until 48 h after. Patients were allowed to swallow liquids 24 h after dilatation.

3. Statistical evaluation

Results are presented as a mean ± S.E.M. Fisher's exact or Student's *t*-test, where appropriate, were used to evaluate statistical significance. Kaplan–Meier curves were used to estimate the probability of re-dilatation need; differences between the two branches were evaluated by means of log-rank test. All the calculations were performed by using the GraphPad Prism 3 running on a Mac computer.

4. Results

Seventy-eight children, all presenting with accidental caustic soda (sodium hydroxide) ingestion, were admitted since December 2005 to May 2007. Clinical characteristics of the patients are summarized in Table 1. All children showed severe strictures often compromising the lumen at different levels. All were circumferential, extending longitudinally for at least 2–4 cm, with redundant pouch-like oesophagus above the stricture in 31 out of 78 patients (i.e. 39.7%). In most of them the stricture was tortuous.

Twenty-five children (group 1) with a mean age of 5 ± 2.5 years had the first dilatation at 37 ± 12 weeks after injury. The long interval from injury and dilatation was due in 14 patients to unavailability of endoscopic and dilatation devices before December 2005. Most of these patients were fed by gastrostomy. The cause of delay in the other children was either a late, slow, insidious onset of dysphagia, which actually led to swallow only liquid or semisolid food, or the temporary unavailability of a surgeon expert in dilatation procedures. In these patients, a gastrostomy was carried out in order either to avoid malnutrition or to facilitate further endoscopic and dilatation manoeuvres. Overall, a gastrostomy was carried out in 16/25 (64%) children. One child was lost to follow-up after the first dilatation to 7 mm. One hundred fifty-four dilatation procedures were carried out in this group (6.4 dilatations/patient), with a mean follow-up of 11 ± 2.5 months. Six dilatation procedures (i.e. 3.8%) were accomplished by a balloon catheter. Savary bougies were used in all other procedures, often in a retrograde fashion in those with gastrostomy. In one child it was not possible to overcome the stricture by the guide wire and after three attempts an oesophageal replacement with stomach was carried out.

As shown in Table 1, the perforation rate in this group was 16.0% (i.e. 4 out of 25 children), with a procedure-related perforation rate of 2.6%. This complication occurred at the first dilatation attempt in three patients and at the third one in the fourth child. Three patients developed subcutaneous emphysema and were treated conservatively. In the fourth patient, an acute upper oesophageal laceration during balloon dilatation (first attempt) was followed by death within few minutes. The death rate in this group was 4.0%.

Dilatation was successful in 22 children (91.6%). All dilated patients resumed good swallowing for solid food. However, 16/22 (72.7%) had a first recurrence of their stricture after few months and 7/22 (31.8%) a second recurrence (Table 1), all requiring a further dilatation, which was equally successful. Compared to group 2, the odds ratio for recurrence was 7.93 (95% C.I. 2.23–28.15).

In group 2, 31 children with a mean age of 5 ± 4 years were observed shortly after injury and submitted to a timely dilatation. The interval between injury and dilatation was 4 ± 1.4 weeks. Twenty-one (67.7%) had a diagnostic endoscopy within 2–3 days after caustic ingestion. In all of them, deep necrotic and transmural damage of the oesophageal wall was found (Fig. 2). A gastrostomy was performed in those with severe oesophageal lesions at early endoscopy, as well as in children complaining of severe dysphagia, even in absence of previous endoscopy. In total, 17/31 patients (54.8%) had a gastrostomy carried out. One hundred forty-one dilatation procedures were performed (4.5 dilatations/patient). Balloon dilatation was used in three patients (2.1%). Only one fatal upper oesophageal laceration using Savary bougienage (death rate: 3.2%) did occur with a procedure-related perforation of 0.7%. A satisfactory dilatation, with a resumed

Table 1
Clinical characteristics and outcome of patients with caustic oesophageal lesions submitted to either delayed or timely dilation

	Group 1: delayed dilation	Group 2: timely dilation	Significance
Number of patients	25	31	NS
Ingestion to dilation interval (weeks)	37 ± 2.4	4 ± 0.25	$p < 0.0001$
Gastrostomy (%)	64	54.8	NS
No. of dilations/patient	6.4	4.5	NS
Follow-up, months (range)	11 ± 2.5 (6–14)	10 ± 2.1 (6–14)	NS
Perforations (%)	16.0	3.2	NS
Procedure-related perforations (%)	2.6	0.7	NS
Death rate (%)	4.0	3.2	NS
Success rate (%)	96.1	96.7	NS
Recurrent stricture (%)	72.7	30	$p < 0.0026$
Second recurrent stricture (%)	31.8	3.3	$p < 0.0231$

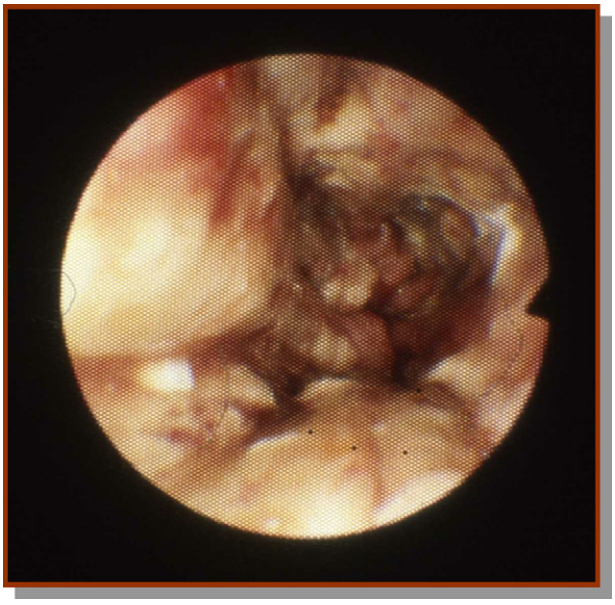

Fig. 2. Deep necrotic and transmural damage of the oesophageal wall 48 h after soda caustic ingestion. Gastrostomy in these patients is mandatory.

good swallowing for solid food, was obtained in 30 out of 31 patients (96.7%). However 9/30 patients (30%) had recurrence of their stricture and 1 (3.3%) had a second recurrence; all were dilated successfully. The follow-up was 10 ± 2.1 months.

The number of perforations was higher in group 1, but the difference fell short of statistical significance. Conversely, the incidences of first and second recurrences were significantly higher in the group that had a late management. The cumulative hazard of re-dilatation in both groups is shown in Fig. 3.

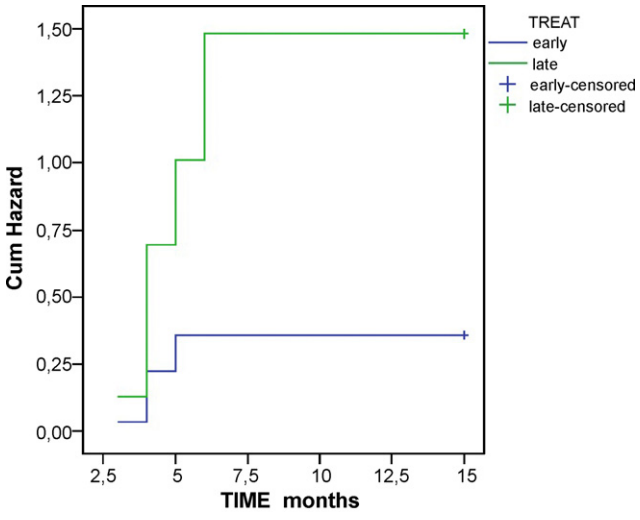

Fig. 3. Cumulative hazard of re-dilatation in patients with oesophageal caustic lesions submitted to early or late dilatation. The difference was significant ($p = 0.0008$) at log-rank test.

5. Discussion

Oesophageal strictures can be morphologically categorized into two groups: simple and complex [2]. Strictures allowing the passage of the endoscope, short, focal and not angulated, can be defined as simple strictures. Conversely, complex strictures are angulated, long (>2 cm), circumferential and with a severely narrowed lumen. Strictures caused by caustic ingestion are usually complex, therefore being most refractory and risky to dilate [2]. Moreover, among corrosive agents, sodium hydroxide (caustic soda) is a very hazardous one [6], producing liquefactive necrosis, full thickness necrosis of the oesophagus and resulting in a noticeable thickness of the wall [7], which, when marked, suggests a transmural damage. Hence it is not surprising that all strictures we found in both groups of reported patients were severe and difficult to dilate, requiring several dilatation attempts.

Reports of outcomes after dilatation therapy for corrosive strictures are scanty, with few studies dealing with a substantial number of patients. Patient's age, stricture location and length, grade of dysphagia at presentation and even early endoscopic findings [5] were not found to be good predictors to response to dilatation.

Understanding the pathophysiology of corrosive injury is important in planning both acute and long-term management. Early dilatation is not recommended and is risky for the fragile oesophageal wall due to persisting inflammation, necrotic tissue sloughing and granulation tissue formation [8]. Scar retraction begins as early as the end of the second week and lasts for 6 months. Six to 12 months is considered the average time before full fibrosis is achieved after injury [9]. The marked fibrosis and collagen deposition during this late period could then make dilatation management more complicated and impair the late clinical outcome.

We have previously reported [10] our initial experience in the management of oesophageal caustic lesions in children, emphasizing that – in this setting – the majority of strictures are late and therefore difficult to dilate and at higher risk of perforation. The aim of this retrospective study was to evaluate the influence, if any, of a delayed *versus* timely dilatation treatment on the clinical outcome of corrosive strictures. Both groups we considered in our study were equally balanced with respect to age, gender, caustic agent and operator, most dilatations having been performed by one of the authors (SC). The strictures we encountered were complex, as expected, and their features did not differ substantially between patients of both groups.

A similar percentage of patients in both groups were submitted to a surgical gastrostomy. The decision for a gastrostomy was mainly dependent on the grade of dysphagia as well as on the complexity of the dilatation. In this setting, the serious nature of oesophageal injury, the children's poor nutritional condition when admitted late after ingestion, together with the impossibility to feed these patients properly with total parenteral or enteral nutrition, makes the discomfort and the inconvenience of a gastrostomy always

worthwhile. Moreover, a retrograde dilatation may be accomplished through the gastrostomy when cannulation of the stricture from above is not feasible, thus making the procedure easier and safer [11,12].

Perforation rate in oesophageal dilatation is reported to be 0.1–0.4% [13,14], but the risk of perforation increases in irregular, longer and high-grade strictures [15], with a reported perforation rate of up to 32% of patients after dilatation of caustic strictures [16,17]. The more severe fibrosis and the reduced compliance of the oesophageal wall, likely present in late dilatations, may be responsible for the higher number of perforations in group 1. However, despite late dilatation carries a fivefold increased risk of perforations, the difference between groups fell short of statistical significance. In three out of five patients, perforation occurred during a balloon dilatation, which was used rarely (nine times) and only during our early experience in this setting. Although some good results have been reported with the use of the balloon [13,18], the failure rate was found to be higher in children with caustic ingestion-related strictures, compared to other benign strictures [19] and the reported perforation rate may be as high as 32% [16]. Therefore, balloon dilatation needs extra caution and has inferior outcomes in caustic strictures.

In two patients (one for each group) an oesophageal laceration during the procedure was followed by a sudden death, likely consequent to a massive pneumothorax with a mediastinal shift. It must then be kept in mind that after corrosive ingestion, the transmural involvement by severe fibrosis of organs adjacent to the upper alimentary tract such as larynx, trachea and aorta may account for an increased risk of severe complications and eventually death. Death rates resulting from perforation are very variable, ranging from 18 to 50% [17,20,21], and treatment delay, may play a role. Indeed, patients referred from other hospitals show a double death rate, compared to those perforated in the same hospital [22].

The success rate, in terms of response to dilatation and of disappearance of dysphagia with return to normal feeding, is high and comparable in both groups. Conversely, first and second recurrences, needing further dilatations, are significantly higher in the group of children who were dilated late after injury. Similar results are reported in the only paper found in the literature focusing specifically on late dilatation after caustic ingestion [3]. Maximal oesophageal wall thickness, as measured by CT scan, but not stricture length, was associated with the number of sessions required for adequate dilatation [7]. Whenever more than one stricture was present, the stricture associated with the greatest maximal oesophageal wall thickness was found to be the most difficult to dilate. Therefore, the depth of fibrosis seems to be the principal determinant of response to dilatation, either in terms of number of sessions or in terms of recurrence. Actually, a more severe fibrosis of the oesophageal wall is very likely when the dilatation is performed late, especially after six months, when full fibrosis and retraction is achieved. Moreover, repeated dilatation procedures can induce themselves a secondary process of fibrosis and scarring. Unfortunately, a late dilatation treatment may be particularly common in low-income countries. The current role of local corticosteroid injection for corrosive oesophageal strictures has not been demonstrated to be conclusively beneficial in corrosive injuries [23,24]. On the contrary, topical application of mitomycin C has been advocated for refractory peptic strictures [25]. Therefore, any effort should be made to treat caustic strictures promptly by adequate dilatation programs.

In conclusion, when corrosive oesophageal strictures are treated late, a more complex procedure and a higher risk of perforation should be expected. A good endoscopic and clinical outcome can be achieved independently of the time of dilatation, but a significantly higher recurrence rate, with consequent need for further dilatation, may be anticipated. These conclusions do not derive from a randomized prospective investigation, which would be unethical and unfeasible. The results of our retrospective study do however suggest that a timely management of the strictures should be strongly pursued.

Practice points

- Accidental ingestion of caustic substances is particularly frequent in children of developing countries.
- In developing countries, the majority of oesophageal caustic strictures in children are usually observed late, when dilatation procedures are likely more difficult and carry a high risk of perforation.
- A good endoscopic and clinical outcome can be achieved independently of the time of dilatation, but a significantly higher recurrence rate is to be expected after late treatment.

Research agenda

- Prevention of stricture development and/or recurrence is a key topic for future research. Topical application of mitomycin C on the oesophageal wall might be valuable in low-income countries, being relatively cheap and not technically difficult.
- Oesophageal replacement surgery is almost unavailable in developing countries, except in very few referral centres. Therefore, dilatation programs should be implemented in district hospitals too, in order to make them widely accessible.

Conflict of interest statement

None declared.

Acknowledgements

We are indebted to the foreign and local doctors and nurses of the "Emergency Surgical Center of Goderich," Freetown. Without their efforts and help this work would have not been possible.

The program of treatment of corrosive injuries in children in Sierra Leone was financially supported by the Fondazione Cassa di Risparmio di Parma.

We are also deeply indebted to Professor George Triadafilopoulos, MD, DSc, FACP, FACG (Stanford University, CA, USA) for his critical reading of the manuscript and the valuable suggestions.

References

[1] Hugh TB, Kelly MD. Corrosive ingestion and the surgeon. J Am Coll Surg 1999;189:508–22.
[2] Lew RJ, Kochmann LM. A review of endoscopic methods of esophageal dilatation. J Clin Gastroenterol 2002;35:117–26.
[3] Gun F, Abbasoglu L, Celik A, Salman FT. Early and late management in caustic ingestion in children. Acta Chir Belg 2007;107:49–52.
[4] Min Keh S, Onyekvelu N, McManus K, McGuigan G. Corrosive injuries to upper gastrointestinal tract: still a major surgical dilemma. World J Gastroenterol 2006;12:5223–8.
[5] Kirsh MM, Ritter F. Caustic ingestion and subsequent damage to the oropharyngeal and digestive passages. Ann Thorac Surg 1976;21:74–82.
[6] Bertinelli A, Hamill J, Mahadevan M, Miles F. Serious injuries from dishwasher powder ingestions in small children. J Pediatr Child Health 2006;42:129–33.
[7] Lahoti D, Broor SL, Basu PP, Gupta A, Sharma R, Pant CS. Corrosive esophageal strictures: predictors of response to endoscopic dilatation. Gastrointest Endosc 1995;41:196–200.
[8] Mamede RC, De Mello Filho FV. Treatment of caustic ingestion an analysis of 239 cases. Dis Esophagus 2002;15:210–3.
[9] Gumaste VV, Dave PB. Ingestion of corrosive substances by adults. Am J Gastroenterol 1992;87:1–5.
[10] Contini S, Tesfaye M, Picone P, Pacchione D, Kuppers B, Zambianchi C, et al. Corrosive esophageal injuries in children. A short-lived experience in Sierra Leone. Int J Pediatr Otorhinolaryngol 2007;71:1597–604.
[11] Anderson KD. Corrosive injury. In: Pearson FG, Cooper JD, Deslauriers J, Ginsberg RJ, Hiebert CA, Patterson GA, Urschel Jr HC, editors. Esophageal surgery. 2nd ed. New York: Churchill Livingstone; 2002. p. 577–89.
[12] Bueno R, Swanson SJ, Jacklitsch MT, Lukanich JM, Mentzer SJ, Sugarbaker DJ. Combined antegrade and retrograde dilatation a new endoscopic technique in the management of complex esophageal obstruction. Gastrointest Endosc 2001;54:368–72.
[13] Hernandez LJ, Jacobson JW, Harris MS. Comparison among the perforation rates of Maloney, balloon and Savary dilation of esophageal strictures. Gastrointest Endosc 2000;51:460–2.
[14] Mandelstam P, Sugawa C, Silvis SE, Nebel OT, Rogers BH. Complications associated with esophagogastroduodenoscopy and with esophageal dilatation. Gastrointest Endosc 1976;23:16–9.
[15] Broor SL, Raju GS, Bose PP, Lahoti D, Ramesh GN, Kumar A, et al. Long term results of endoscopic dilatation for corrosive esophageal strictures. Gut 1993;34:1498–501.
[16] Song H, Han Y, Kim HN, Kim CS, Choi KC. Corrosive esophageal stricture: safety and effectiveness of balloon dilatation. Radiology 1992;184:373–8.
[17] Karnak I, Tanyel FC, Buyukpamukcu N, Hicsonmez A. Esophageal perforations encountered during the dilation of caustic esophageal strictures. J Cardiovasc Surg 1998;39:373–7.
[18] Mekki M, Said M, Belghith M, Krichene I, Chelly S, Jouini R, et al. Pneumatic dilatation of caustic esophageal stenosis in children. Report of five cases. Arch Pediatr 2001;8:489–92.
[19] Sandgren K, Malmfors G. Balloon dilatation of esophageal strictures in children. Eur J Pediatr Surg 1998;8:9–11.
[20] Avanoglu A, Ergun O, Mutaf O. Management of instrumental perforations of the esophagus occurring during treatment of corrosive strictures. J Pediatr Surg 1998;33:1393–5.
[21] Guitron A, Adalid R, Nares J, Mena G, Gutierrez JA, Olivares C. Benign esophageal strictures in toddlers and pre-school children. Results of endoscopic dilatation. Rev Gastroenterol Mex 1999;64:12–5.
[22] White RK, Morris DM. Diagnosis and management of esophageal perforations. Am Surg 1992;58:112–9.
[23] Pelclová D, Navrátil T. Do corticosteroids prevent oesophageal stricture after corrosive ingestion? Toxicol Rev 2005;24:125–9.
[24] 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. Clin Toxicol (Phila) 2007;45:402–8.
[25] Uhlen S, Fayoux P, Vachin F, Guimber D, Gottrand F, Turck D, et al. Mitomycin C: an alternative conservative treatment for refractory esophageal stricture in children? Endoscopy 2006;38:404–7.