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Early and Late Term Management in Caustic Ingestion in Children : A 16-year Experience

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Abstract. Accidental corrosive ingestion is one of the common problems causing serious esophageal strictures in children. The acute phase treatment has a great effect on stricture development. In this study we aim to present our experience in the management of caustic ingestion, particularly during the acute phase. From January 1990 to January 2005, 296 children were admitted to our clinic with caustic ingestion. Ninety-one patients who received dilatation treatment due to esophageal strictures constituted the present study group. Forty-three of them were admitted to our centre immediately after caustic ingestion (Group A) whereas 48 of them received some kind of treatment in other hospitals and were referred us with the diagnosis of stricture 6 to 12 weeks after ingestion (Group B). In the acute phase, the patients were given nothing orally until esophagoscopy was performed in the first 24-48 hours. The patients with grades 2b and 3 lesions underwent a week of esophageal rest by using a nasogastric tube. IV fluids and broad-spectrum antibiotics with a single-dose steroid were given. IV ranitidine was also added to the medical treatment. If there were stricture formations on barium meal after 3 weeks, these patients underwent esophageal dilatation programmes. The response rates to dilatation treatment were higher in group A. In addition, increased perforation rates were observed in group B. Sixty per cent of patients in group A but none of the patients in group B have recovered in the first year.
In conclusion, after caustic ingestion, esophageal rest combined with supporting treatment seems to provide a good success rate with respect to prevention of stricture development and other troublesome complications.

Introduction

Corrosive ingestion in children, may cause clinical manifestations varying from no injury to fatal outcome, and one of the important complications is esophageal stricture following esophagitis (1). The optimal treatment of corrosive ingestion remains controversial. Acute management and dealing with the complications differ according to the condition of the patient and the policy of the centre (2, 3).

In this study, we aim to present the treatment results of our series divided into two groups with esophageal strictures as follows ; the patients in the first group treated immediately after caustic substance ingestion whereas the patients in the second group were referred us from other hospitals after 6-12 weeks of the ingestion.

Patients and Methods

From January 1990 to January 2005, 296 children were admitted to our clinic with caustic ingestion. All ingestions were reported to be accidental and the etiologic agent was NaOH in 76% of patients. Ninety-one patients who received dilatation treatment due to esophageal strictures constituted the present study group. Forty-three of them were admitted to our centre immediately after caustic ingestion, having developed stricture afterwards (Group A) whereas 48 of them had received some kind of treatment after initial esophagoscopy in different hospitals and were referred us with the diagnosis of stricture 6 to 12 weeks after ingestion (Group B).
After acute ingestion, our management protocol was as follows : the type of substance ingested, physical examination findings including the condition of perioral skin and oral mucosa, and the ability to swallow saliva were recorded. The patients were given nothing orally until esophagoscopy which was performed in the first 24-48 hours. In all cases, endoscopic examinations were performed under general anaesthesia by rigid esophagoscopy. IV fluids and broad-spectrum antibiotics with a single-dose steroid were given. IV ranitidine was also added to the medical treatment. The grade of esophageal burn during endoscopy was determined in a scale ranging from zero to three, as previously described (4).

Patients with grades 1 and 2a lesions were fed orally, discharged and called for a barium esophagogram after three weeks. The patients with grades 2b and 3 lesions were fed enterally via a soft nasogastric tube for one week with the aim of preventing the contact of food particles with esophageal mucosa. Thereafter the tube was

removed and oral feedings were started. These patients were examined for esophageal stricture with an esophagogram three weeks after ingestion. If there were stricture formations on barium meal with or without swallowing difficulties, these patients underwent esophageal dilatation programmes. The dilatations were performed every three weeks during the first three months after which the programme was rearranged according to the responses.

The dilatations were performed with Tucker, Rusch or balloon dilatators under general anaesthesia. Patients with an esophageal perforation owing to dilatation, with intractable strictures or with an irregular shaped esophagus, underwent a gastrostomy, and the dilatations were made in a retrograde fashion with Tucker dilatators, that carry a lower perforation risk. If available, balloon dilatators were used for strictures with low luminal diameters, which were potentially susceptible to perforation. In the rest of the patients, Rusch dilalators were used in antegrade fashion.

Our approach to the patients admitted to our clinic, the differences between the two subgroups (A and B) in the first endoscopic evaluation, the stricture lengths, the luminal diameters at the stricture zones, the route of the dilatations, the responses to the dilatations, the complication (perforation, mediastinitis, lung abscess, sepsis), and survival rates were evaluated.

Results

Group A : Patients with acute caustic ingestion who were admitted to our institution and, after the determination of the esophageal stricture, involved in an early dilatation programme on the third week according to our protocol.

Group B : Patients who were referred to our institution by other healthcare givers 6-12 weeks after caustic ingestion, who underwent acute phase esophagoscopy and then received different treatment protocols that may have included null or inappropriate protocols.

Group A consisted of 43 patients (26 boys and 17 girls, with a mean age 4) and group B consisted of 48 patients (30 boys and 18 girls, with a mean age 5) ; amounting to 91 patients with esophageal strictures who underwent the dilatation programme. Thirty-six of the 43 and 38 of the 48 had a history of alkali substance ingestion. The grades of initial esophageal burns and stricture lengths are shown in Tables I and II.

When the luminal diameters of the strictures of the groups were studied, the rate of 0-3 mm for diameters was higher in group B, and 7-10 mm for diameters was higher in group A. On the other hand, the response rates to dilatations were higher for all strictural diameter categories in group A. In addition, an increased perforation rate was observed in group B (Table III).

Table I

Comparison of the groups according to the grades of the esophageal burns

Grades	Group A		Group B	
	n	%	n	%
2a	7	16.2	4	8.3
2b	28	65.1	32	66.6
3	8	18.6	12	25
Total	43		48	

Table II

The stricture lengths of the patients

Stricture lengths	Group A Patient no	%	Group B Patient no	%
3-5 cm	12	27.9	13	27.0
6-9 cm	14	32.5	16	33.3
>10 cm	17	39.5	19	39.6

Sixty per cent of the patients in group A but none of the patients in group B have recovered in the first year (Table IV).

As shown in Table V, complication rates were lower in group A. In addition, Table VI summarizes the final conditions, further treatment modalities and mortality rates.

Discussion

Accidental corrosive ingestion is one of the common problems in children worldwide (5). Recently, in Turkey, as uncontrolled and cheaper domestic cleaners have been introduced through common informal or open markets, the caustic ingestion type of household accident in children has begun to increase, particularly amongst families with low income. The degree and the extent of the corrosive lesion depend on several factors as follows ; the characteristics of the caustic substance (concentration and pH), the quantity swallowed, the duration of contact with the tissues, and the fullness of the stomach. On the other hand, the development of the strictures is mainly related to the depth and circumferential nature of the injury (6). Because there is no protective eschar formation in alkali burns, the depth of penetration caused by liquefactive necrosis is much greater, in comparison with acidic burns (7). In our series, most caustic ingestions were caused by alkali, as in other series (8-10). Unfortunately, in Turkey, these caustics, particularly the alkali agents as drain cleaners and oil solvents are sold without childproof containers, generally in open markets.

Table III

Differences of groups in luminal diameters at stricture levels, response rates to dilatations, and perforation rates

Luminal diameters	Patient no A	%	B	%	Succesful treatment results A	%	B	%	Perforation A	%	B	%
0-3 mm	5	11.6	20	41.7	1	20	1	3.8	4	80	15	75
4-6 mm	16	37.2	15	31.2	6	37.5	4	26.6	5	31.2	9	60
7-10 mm	22	51.2	13	27.1	21	95.4	7	53.8	–	–	–	–
Total	43	100	48	100	28	65.1	12	25	9	20.9	24	50

Table IV

Dilatation periods of the groups

Dilatation periods	Group A Patient no	%	Group B Patient no	%
0-12 months	17	60.7	–	–
12-24 months	10	35.8	5	41.7
24-36 months	1	3.5	5	41.7
> 36 months	–	–	2	16.6
Total	28	100	12	100

Table V

Complications

Complications	Group A Patient no	Group B Patient no
Perforation	9	24
Mediastinit	7	12
Abscess of lung	2	4
Empyema	8	20
Pericardial effusion	2	4
Sepsis	1	3
Mortality	–	3

Table VI

Additional treatment modalities and survival rates

Modality	Group A No :	%	Group B No :	%
Cured with dilatations	28	(65.1)	12	(25)
Patients still on dilatation programme	15	(34.9)	24	(50)
Colonic transposition	–		3	(6.25)
Patients with stents	–		4	(8.4)
Mortalitiy	–		3	(6.25)
Total	43	(100)	48	(100)

Although the optimal management of corrosive ingestion has not been well proposed, the rate of complications, particularly strictures, and the outcome mainly depend on the initial management and care. Several management protocols have been recommended, including resting the esophagus or the whole of the gastrointestinal tract, the use of broad-spectrum antibiotics, systemic steroids, antacids and H_2 receptor blockers. The resting of the esophagus is either achieved by using a nasogastric tube or gastrostomy, whereas the whole gastro-intestinal tract is allowed to rest with total parenteral nutrition (2, 11, 12).

Acute phase management should be directed at preventing esophageal strictures which have the potential to make the rest of the victim's life unbearable.

Our main policy is to give nothing orally before esophagoscopy, which was performed in the first 24-48 hours. We used a rigid esophagoscope because flexible devices are not available in our department. Patients with mild lesions are fed orally, told to come for a barium esophagram after three weeks, even if there are no problems, and then discharged. None of them required further treatment. The patients with more serious lesions were fed enterally via a soft nasogastric tube for a week with the aim of preventing the contact of food with esophageal mucosa even if the child could swallow. Because, to our knowledge, an infection which may be caused by bacterial colonization owing to a food impaction, is one of the factors influencing the inflammatory reaction in the first few days and the collagen synthesis may later play a role in future stricture development (2, 12). From this point of view, the tube was removed and oral feeding was allowed a week after the collagen production began to decrease.

During the first week, IV fluids and broad-spectrum antibiotics – also covering the oral anaerobics – were given with a single dose steroid to decrease the oedema ; however there was no measurable benefit from the use of steroids when compared with those patients who did not receive steroid treatment (9). In addition, IV ranitidine was used with the aim of decreasing the secondary effects of possible acidic reflux.

Although the grade of esophageal burns and stricture lengths were similar in groups A and B, the response to dilatation treatment was higher in group A and esophageal perforation rates were higher in group B. This case suggests the importance and effectiveness of early correct treatment as explained above, which seems quite effective for the acute phase for the present. In

addition, dilatation programme periods, complication rates, the need for further treatment modalities, and finally the mortality rate between two subgroups, indicate the superiority of our treatment protocol for acute caustic ingestion terms, although the two groups were not entirely comparable.

In conclusion, the main goal in acute phase treatment should be saving the esophagus from inflammatory reaction and to achieve appropriate wound healing with the aim of having an acceptable esophageal anatomy for a better quality of life. Certainly this statement should be supported by prospective trials, preferably by using histological parameters.

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