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Predictive value of visible lesions (cheeks, lips, oropharynx) in suspected caustic ingestion: May endoscopy reasonably be omitted in completely negative pediatric patients?

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The relationship between absence or presence of grossly visible lesions in the cheeks, lips, and oropharynx (C.L.O. burns) and the incidence, site, and degree of visceral burns was evaluated in all children referred to our hospital for a suspected caustic ingestion during a 10-year period. All children underwent eso-gastro-duodenoscopy within 24 hours. Of the 156 children, 96 (61.6%) showed no visible signs of contact with the caustic substance; however, in 36/96 (37.5%), endoscopy revealed burns in one or more visceral sites. Eight of 36 children (22.2%) sustained potentially dangerous lesions (second to third degree).

Sixty of 156 children (38.4%) showed visible lesions; in 30/60 (50%), endoscopy revealed other burns in one or more visceral sites. Fourteen of 30 patients (46.6%) sustained potentially dangerous lesions (second to third degree).

A total of 50 esophageal burns have been recorded: first degree (E1), 32; second degree (E2), 12; third degree (E3), 6. Two of 12 patients with E2 lesions and 6/6 with E3 lesions developed esophageal stenosis. One patient in this latter group died because of complications related to a tracheostomy.

A total of 31 gastric burns have been recorded: G1 (22), G2 (6), G3 (3). One gastric perforation was observed in the G3 group, whereas the remaining two lesions healed with residual asymptomatic scarring. Minimal scarring was observed in two of six patients with G2 burns.

A total of eight lesions have been recorded in the larynx [L1 (3), L3 (1)] and in the duodenum [D1 (2), D2 (2)].

Analysis of our data shows that: 1. Absence of visible lesions cannot exclude the occurrence of visceral burns. 2. In the patients showing C.L.O. lesions, the risk of dangerous visceral burns (second to third degree) is higher than in those without C.L.O. lesions. 3. Higher than first degree C.L.O. lesions almost invariably are associated with dangerous visceral burns. 4. Signs or symptoms do not adequately predict the presence or the severity of visceral lesions, even if spontaneous vomiting is associated with a high incidence of second to third degree visceral burns.

Eso-gastro-duodenoscopy, with extremely wide indications, is mandatory in all children referred for obvious or suspected caustic ingestion.

There are many dangerous substances on the market today. Household products usually contain harmful substances, often toxic or caustic elements. Not uncommonly, the effective composition of the product is not clearly explained or is completely lacking on the package. Moreover, the containers can easily be opened by small children.

A quick investigation carried out by the author (C.P.) in his own home revealed that of a total of 31 different home products, 13 were caustic, eight were toxic, and six were both caustic and toxic. In only one case was correct information concerning the chemical composition and its relative danger given by the manufacturer, and insufficient instructions were given on what to do in case of accidental ingestion in only one case. Only two containers (7%) were child safe.

People tend to feel safe with home products because the mass media humanizes these products by means of skillful advertising. Unconscious of the dangers, parents do not take enough care in keeping the containers out of the reach of children. As a result, children come into contact with these substances and in many instances suffer the consequences.

The role of endoscopy in the diagnosis and immediate treatment of acute lesions and late complications has been stressed in the literature.[1-8] The limits of the rigid esophagoscope, which is used mainly by otolaryngologists, are well known; the investigation should be performed only to the level of the first circumferential severe burn. Consequently, other severe injuries may be missed.[9-11]

The major problem for the physician is to resist the temptation to not do the endoscopy in children with no visible lesions and with no signs or symptoms. On the other hand, the parents also tend to draw back in these cases. Signs and symptoms do not adequately predict the presence or the severity of an esophageal lesion, as stated by Gaudreault et al.[3]; in particular, many authors have outlined that the ab-

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sence of oropharyngeal lesions does not exclude the presence of esophageal or gastric injury,[1-4,10] even if Ament et al.[5] recently stressed that patients free of burns in the lips, buccal mucosa, or oropharynx rarely have a significant burn in the esophageal or gastric mucosa. This study was undertaken to evaluate the relationship between the absence or presence of grossly visible lesions and the incidence, site, and degree of visceral burns in all children referred to our hospital for obvious or suspected caustic ingestion.

METHOD

From May 1978 to December 1988, all children coming to the General Hospital of Padua, Italy for suspected, probable, or obvious caustic ingestion underwent initial evaluation in the Pediatric Medical Department. All patients were then referred to our Pediatric Surgical Department for endoscopy and further treatment; almost all endoscopies were performed by the author (C.P.). The Pediatric Emergency Care Unit, in this department, received the patients who showed life-threatening symptoms.

These patients accounted for the total pediatric population who were treated for caustic ingestion in our town.

After the initial evaluation (general examination, blood analysis, x-rays of thorax and abdomen) all children were submitted to eso-gastro-duodenoscopy within 24 hours, whatever the external appearance and symptomatology. In children less than five years of age, who represent 91.3% of this group, the cries which followed the moment of the probable accident were considered a sufficient criterion to warrant endoscopy.

The following data were recorded: 1) chemical property of the caustic substance (acid or basic); commercial name; 2) age and sex of the child; 3) actual symptoms (salivation, disphagia, abdominal pain, respiratory distress); 4) absence or presence of visible C.L.O. lesions; 5) timing and modality of the accident, presence of spontaneous vomiting and 6) presence and degree of visceral burns, as demonstrated by fibroendoscopy, in the larynx (L), esophagus (E), stomach (S) and duodenum (D).

The lesions were classified and recorded according to Kirsch et al.[12] and were commonly reported in literature[3,13] as follows: 1) first degree burn: limited to the mucosa, presence of edema or erythema; 2) second degree burn: penetrating beyond the mucosa, presence of ulceration or whitish membrane; 3) third degree burn: transmural involvement of the visceral wall, eg G2 = second degree burn of the stomach. Data were finally analyzed using the Chi-square test and its Yates correction.

RESULTS

One hundred fifty-six children, 90 males (57.6%) and 66 females (42.4%), with a mean age of 33.4 months (min = 1 month, max = 11.5 years), were referred to our department for probable or suspected caustic ingestion. 73.7% of the patients were less than three years of age. Four patients (2.5%) were treated at the Emergency Care Unit for dyspnea (three cases) or shock (one case).

All children underwent eso-gastro-duodenoscopy within 24 hours; a flexible fiberoptic pediatric instrument (Olympus GIF-P2, XP-10, XP-20) was used in all cases.

The substance was identified as acid, basic, and mixed in 12.8%, 86.5%, and 0.6% of the cases, respectively. Among basic substances, the most frequently involved were sodium hypochloride (40%), sodium hydroxide in powder or liquid form (29.6%), and ammonia (17.7%).

No difference between males and females was found concerning age. The majority of the children showed no specific physical signs other than visible lesions. A record of actual symptoms was prepared, but ultimately was considered unreliable; in fact, the low average age of the population (33.4 months) made the evaluation of symptoms such as disphagia very difficult, whereas refusal to drink was in most cases the consequence of even minimal oropharyngeal lesions or a natural reactive behavior of the child.

The finding of spontaneous or provoked abdominal pain was bizarre. 12.8% of the patients showed spontaneous vomiting soon after the accident. As demonstrated by Gaudreault,[3] if considered alone it is associated with 33% of second to third degree burns. The incidence of these lesions in the corresponding group of our series accounts for 40% and increases to 75% in the 10 patients with associated visible lesions.

Ninety-six of 156 children (61.8%) showed no visible signs of contact with the caustic substance; however, in 36 of 96 (37.5%), endoscopy revealed burns in one or more visceral sites. Table 1 shows the distribution of the lesions among these patients.

A total of 48 burns were recorded, with an average of 1.33 for each patient. Eight of 36 children (22.2%) sustained potentially dangerous lesions (≥second degree). The remaining 60 children with no visible lesions were completely negative. Sixty of 156 patients (38.4%) showed evident C.L.O. burns; the distribution is reported in Table 2.

Among the 53 children with C.L.O.-first degree burns, 16 (30%) showed other lesions of the same degree, and seven (13.2%) showed one or more visceral burns of a higher degree. All seven patients with C.L.O. burns ≥second degree suffered from one or more second and third degree visceral lesions. Thirty of 60 children (50%) with C.L.O. burns were proved to have other burns in one or more visceral sites; the distribution of these lesions is reported in Table 3.

A total of 41 different burns were found, with an average of 1.36 for each patient. Fourteen of 30 patients (46.6%) sustained potentially dangerous lesions (≥second degree). The incidence of significantly visceral burns has been 8.3% (8/96)

TABLE 1
Distribution of the lesions among 36/96 children without C.L.O. burns

Single site: 25/36 (69.4%)	L1 = 1/36 (2.7%)
	E1 = 12/36 (33.3%)
	E2 = 1/36 (2.7%)
	E3 = 2/36 (5.4%)
	G1 = 7/36 (19.4%)
	G2 = 1/36 (2.7%)
	D1 = 1/36 (2.7%)
Multiple sites: 11/36 (30.5%)	E1-G1 = 6/36 (16.6%)
	E1-G2 = 2/36 (5.4%)
	E1-G1-D2 = 1/36 (2.7%)
	E2-G1 = 2/36 (5.4%)

TABLE 2
Distribution of C.L.O. burns

C.L.O. 1st degree = 53/60 (88.3%)
C.L.O. 2nd degree = 6/60 (10.0%)
C.L.O. 3rd degree = 1/60 (1.6%)

TABLE 3
Distribution of the lesions among 30/60 children with C.L.O. burns

Single site: 22/30 (73.3%)	E1 = 9/30 (30.0%)
	E2 = 6/30 (20.0%)
	E3 = 1/30 (3.3%)
	G1 = 5/30 (16.6%)
	G2 = 1/30 (3.3%)
Multiple sites: 8/30 (26.6%)	L1-E1 = 1/30 (3.3%)
	L1-E2-G3 = 1/30 (3.3%)
	L3-E3 = 1/30 (3.3%)
	E1-G1 = 1/30 (3.3%)
	E2-G2-D1 = 1/30 (3.3%)
	E2-G2-D2 = 1/30 (3.3%)
	E3-G3 = 2/30 (6.6%)

and 23.3% (14/60) in the group with or without C.L.O. lesions, respectively.

A total of 50 esophageal burns have been recorded (E1 = 32, E2 = 12, E3 = 6). Only one of the 32 patients with E1 lesions was treated with steroids for an acute laryngeal edema following ammonia ingestion. The remaining received local treatment for C.L.O. burns, when necessary, and were discharged soon after endoscopy. Twenty-one of them (65.5%) did not show C.L.O. lesions.

All but one of the 12 patients showing E2 burns were treated with antibiotics for 10 days and steroids for at least 21 days; three of them (25%) were effectively free of C.L.O. burns. One child needed total parenteral nutrition and gastrostomy for a deep gastric lesion. Two of 12 patients (16.5%) developed late esophageal stenosis.

All six patients with E3 burns were treated with antibiotics and steroids; two of them (33%) were effectively free of C.L.O. burns. One patient suffered a sudden gastric perforation on the fifth day of admission, which was treated by means of limited gastric wall resection and gastrostomy. One patient needed a tracheostomy for laryngeal damage and a gastrostomy for further retrograde dilatation. Unfortunately, he died because of complications related to the tracheostomy, when the dilatation program for esophageal stenosis seemed to be working. In spite of medical therapy and supportive total parenteral nutrition (three cases) all six children with E3 burns developed esophageal stenosis after discontinuation of steroid therapy.

Early treatment by means of dilatations was started in seven patients with symptomatic esophageal stenosis; today the seven survivors show a normal lifestyle with no dietary restrictions and have not undergone dilatations in the last 12 months.

A total of 31 gastric burns have been recorded (G1 = 22, G2 = 6, G3 = 3). The aforementioned perforation was observed in G3 group, whereas the remaining two lesions healed with residual asymptomatic scarring. Minimal scarring was also observed in two of six patients with G2 burns.

A total of eight other lesions have been recorded in the larynx (L1 = 3, L3 = 1) and in the duodenum (D1 = 2, D2 = 2). The L3 burn required a tracheostomy, whereas the other lesions healed with no further problems.

Routine endoscopy was therefore proven to be helpful in all the cases to ensure the most appropriate treatment, mainly in those patients with second or third degree burns who were free from C.L.O. lesions (5/18 = 27%).

We do not wish to draw any conclusions about the role of therapy with antibiotics and steroids in preventing esophageal stenosis, although it seems probable that this therapy may be effective in the treatment of second degree burns.

As far as the main purpose of this paper is concerned, we can conclude that the statistical analysis of our data shows the following: 1) Absence of visible lesions cannot exclude the occurrence of visceral burns, even if in this case the risk of potentially dangerous lesions remains low (8.3%). 2) In the patients showing C.L.O. lesions, the likelihood of visceral burns is significantly higher than in patients without C.L.O. lesions (χ^2 test, $P \leq 0.05$). 3) In the patients with C.L.O. lesions, the risk of dangerous visceral burns (≥second degree) is also higher than in those without C.L.O. lesions (χ^2 test, $0.05 < P < 0.1$). 4) The simple finding of C.L.O. burns does not enhance the risk of multiple visceral lesions. 5) When C.L.O. burns show a mild degree (first), the risk of other lesions of a higher degree is relatively low (13.2%); higher than first degree C.L.O. lesions almost invariably are associated with potentially dangerous visceral burns.

It has been stressed in the literature[1-8,10] that the absence of any visible burn or pharyngoesophageal symptoms cannot predict the absence of visceral lesions; this was confirmed by Gaudreault et al.,[3] who stated on the other hand that vomiting is associated with a remarkable proportion of second to third degree esophageal lesions. Our data agree with both considerations. On the other hand, our data do not agree with other aspects of their study; in fact, the incidence of first-second-third degree visceral lesions is quite different whether or not patients have external signs, as well as the risk of early complications or late sequelae.

Caustic ingestion remains a frequent accident, at least in our country. Progress in the production of child-safe caps has been slow, and the relevant laws have not yet been passed. Early eso-gastro-duodenoscopy is mandatory in all children referred for suspected, probable, or obvious caustic ingestion.

REFERENCES

1. Haller JA, Andrews GH, White JJ, et al. Pathophysiology and management of acute corrosive burns of the esophagus: Results of treatment in 285 children. J Pediatr Surg 1971;6:578–585.
2. Cello JP, Fogel RP, Boland R. Liquid caustic ingestion. Spectrum of injury. Arch Intern Med 1980;140:501–504.
3. Gaudreault P, Parent M, McGuigan MA, et al. Predictability of esophageal injury from signs and symptoms: A study of caustic ingestion in 378 children. Pediatrics 1983;71:767–770.
4. Rothstein FC. Caustic injuries to the esophagus in children. Pediatr Clin North Am 1986;33:665–674.
5. Ament ME, Berquist WE, Vargas J, et al. Fiberoptic upper intestinal endoscopy in infants and children. Pediatr Clin North Am 1988;35:141–155.
6. Moazam F, Talbert JL, Miller D, et al. Caustic ingestion and its sequelae in children. South Med J 1987;80:187–190.
7. Edmonson MB. Caustic alkali ingestions by farm children. Pediatrics 1987;79:413–416.
8. Waldron DJ, Fitzgerald RJ. Corrosive injury of the esophagus in childhood. Scand J Gastroenterol 1988;23:847–850.
9. Puhakka HJ. Esophageal ulceration due to oral medications. ORL 1987;49:321–326.
10. Sellars SL, Spence RAJ. Chemical burns of the oesophagus. J Laryngol Otol 1987;101:1211–1213.
11. Thompson JN. Corrosive esophageal injuries. I. A study of nine cases of concurrent accidental ingestion. Laryngoscope 1987;1060–1068.
12. Kirsch MM, Peterson A, Brown JW, et al. Treatment of caustic injuries of the esophagus. A ten-year experience. Ann Surg 1977;188:675–678.
13. Hawkins DB, Demeter MJ, Barnett TE. Caustic ingestion: Controversies in management: A review of 214 cases. Laryngoscope 1987;97:1060–1068.