New therapeutic approach to corrosive burns of the upper gastrointestinal tract

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SUMMARY The therapeutic approach to the management of corrosive burns of the upper gastrointestinal tract leaves a considerable morbidity and a heavy mortality rate. This work evaluates the effectiveness of a new therapeutic approach given to 94 consecutive patients. The management has been based on three major points: (1) the definition of extent of upper gastrointestinal lesions by immediate fibroendoscopy; (2) immediate protection of the upper gastrointestinal tract by total parenteral nutrition in cases with serious burns (41 cases), normal oral nutrition being allowed for minor burns (35 cases); (3) reparative surgical procedures for any of the sequelae of such burns during the fibrosing phase. The results were as follows: (a) healing, depending upon the degree of burn, occurred between eight to 90 days; (b) the frequency of subsequent local complications was small with total parenteral nutrition started after a few hours after ingestion of the corrosive product; (c) after reconstructive surgery no serious complications occurred; (d) the overall mortality stayed at a very low level (4 patients). We conclude that the general prognosis of a severe burn of the upper gastrointestinal tract, without other trauma, is appreciably improved by the very early institution of total parenteral nutrition.

Corrosive burns of the upper gastrointestinal tract still remain a very serious condition. The various management regimens attempted hitherto do not appear to have materially improved the prognosis of such cases.1 The aim of this study is (1) to define a management protocol which we have applied over the past four years and (2) to analyse the results and suggest a co-ordinated approach which could ultimately form the basis of a controlled trial if necessary.

Methods

PATIENTS

Over the past four years 94 patients have been referred after ingestion of corrosive products. There were 70 females and 24 males with an age range of 15–86 years with a mean age of 40 years. In 75 cases ingestion of the corrosive was due to a self-poisoning act and in the remainder was accidental. Admission to hospital occurred in 93 cases between one and 48 hours after ingestion and in one case after eight days. In 46 cases, L’Eau de Javel (bleach agent with sodium hydrochloride) was the agent used; in 21 cases acid products were swallowed, 19 of which contained hydrochloric acid, in one case nitric acid, in another a mixture of hydrofluoric and oxalic acid, and in two cases sulphuric acid. In 27 cases alcalis were ingested, 25 with concentrated caustic soda and two with ammonia.

THERAPEUTIC METHODS

Oesophagastroduodenoscopy (Olympus GIF P) was undertaken by the same operator in all cases as soon as the patient was admitted. This proved practical in 81 cases between the first and ninth hour after ingestion of the corrosive (maximum frequency between the second and fourth hours), in 12 cases between 12 and 48 hours, and in one case after eight days.

Whenever possible no formal premedication or sedative was used before fibroscopy and in no case was local oropharyngeal anaesthesia required. An otolaryngological (ORL) examination always preceded fibroscopy. The introduction and the progression of the fibroscope was always performed under visual control and retrovisual methods were not attempted. Depending upon the results of the
endoscopic examination, the mucosal burns were graded in three stages: stage I: simple inflammation; stage II: a few ulcerations, focal necrosis limited to a part of the oesophagus and/or the stomach, slight haemorrhage; stage III: multiple ulcerations, extensive necrosis involving all the oesophagus, and/or the stomach, massive haemorrhage with haematemesis.

As soon as immediate resuscitative procedures had been completed total parenteral nutrition (TPN) was the rule for patients with burns of stages II and III. The nutritive mixtures were never used routinely except for very short periods (a few days) in cases with laryngeal oedema (four patients). Antibiotics were used only in patients with obvious infections and then only where the infecting organism was identified. In no cases were nasogastric tubes left in place or bouginages carried out.

OBSERVATION PROTOCOL
A contrast radiological examination of the upper gastrointestinal tract with an iodide contrast medium was arbitrarily undertaken on the fifteenth day and thereafter monthly until four months after the initial incident.

A control fibroendoscopic examination was always undertaken immediately in all patients after the upper gastrointestinal contrast studies until four months after the initial incident.

When healing of the mucosa had been confirmed by endoscopy the final examination comprised an upper gastrointestinal barium evaluation and radiocinematography to determine the kinetics of the passage of Killian, obviating final stenosis at the cardia of the stomach.

The treatment of eventual consequences of corrosive ingestion was managed surgically in the healing phase. Normal alimentation could be restarted 15 days after such intervention. In the absence of any sequelae normal oral nutrition was restarted immediately after total parenteral nutrition. The patients were followed up for several months (48 months maximum) with radio-cinematography and a final endoscopic examination.

Results

LESIONS OF UPPER GASTROINTESTINAL TRACT
Lesions observed at first endoscopic examination
Frequency Early endoscopy afforded confirmation of upper gastrointestinal tract burns in 76 out of 94 cases.
Location (Table 1) Fibroendoscopy revealed gastric involvement in 70 out of 76 cases. The gastric lesions were associated with an oesophageal burn in 40 cases and were isolated in the other 30. The relationship to the product ingested was that acids and alkalis involved the oesophagus and stomach simultaneously in 23 out of 34 cases, whereas concentrated bleach frequently involved the stomach alone (25 cases out of 42). A comparison of lesions localised to the oropharynlarypeal area and the upper gastrointestinal tract (Table 2) showed involvement of the upper gastrointestinal tract in 46 cases with no involvement of the oropharyngeal area. In contrast, involvement of the oropharyngeal area was accompanied in 31 out of 32 cases by involvement of the upper gastrointestinal tract and the relationship did not seem to be due to the severity of the burn lesions.

Table 2 Comparison of corrosive burns of oropharynlarypeal area and of upper gastrointestinal tract

<table>
<thead>
<tr>
<th>Stage and no.</th>
<th>Upper gastrointestinal tract injury (stage and no.)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>O</td>
</tr>
<tr>
<td>ORL injury</td>
<td></td>
</tr>
<tr>
<td>0: 62</td>
<td>16</td>
</tr>
<tr>
<td>I: 19</td>
<td>0</td>
</tr>
<tr>
<td>II: 6</td>
<td>1</td>
</tr>
<tr>
<td>III: 7</td>
<td>1</td>
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</table>

Stage 0: absence of lesion.

Severity (Table 3) The maximum severity of the lesions observed by fibroendoscopy in the oesophagus or in the stomach were 35 cases stage I, 27 cases stage II, and 10 cases stage III. Thus there were 41 severe or very severe burns in 76 cases with upper gastrointestinal involvement. Concentrated bleach or alkalis were responsible for almost half the serious burns (stage II), whereas the very serious
Table 3  Relationship of product ingested to intensity of lesions observed in upper gastrointestinal tract during acute period

<table>
<thead>
<tr>
<th>Agent</th>
<th>No.</th>
<th>Stage</th>
<th></th>
<th></th>
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<th></th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>I</td>
<td>II</td>
<td>III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium hypochloride</td>
<td>42</td>
<td>21</td>
<td>7</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acids</td>
<td>12</td>
<td>4</td>
<td>1</td>
<td>7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alkalis</td>
<td>22</td>
<td>10</td>
<td>9</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>76</td>
<td>35</td>
<td>27</td>
<td>14</td>
<td></td>
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</tbody>
</table>

burns (stage III) seemed to be more related to acid ingestion.

With concentrated bleach the more serious lesions were observed after ingestion of 300 ml or more, though small amounts of the order of 50 ml were capable of causing stage II lesions. With acids and alkalis widespread destructive lesions were seen only after large quantities of concentrated products had been ingested, though a single mouthful could on occasion produce serious lesions.

Irrespective of the type of product ingested no noticeable differences were apparent with respect to the severity of the corrosive burns in relationship to time of contact. The maximum severity of the lesions occurred before the second hour after ingestion.

Severity related to the nature (self-poisoning or accidental) of the poisoning in the 94 cases revealed that, in 85 cases of self-poisoning, one-half of the patients showed stages II and III lesions. In 19 cases of accidental ingestion, one-third of the patients showed stages II and III lesions.

Associated problems Spontaneous, retrosternal, or epigastric pain was occasioned in four cases (two stage II and two stage III). Moderate pain on epigastric palpation was found in almost all the patients who had ingested a corrosive product and the associated lesions varied between stage I and II.

Laryngeal oedema requiring a short course of steroid therapy occurred in three cases after acid ingestion, and in two after caustic soda. Inhalation bronchopneumonia occurred in two patients after ingestion of hydrochloric acid.

Lesions observed at radiological examinations The radiological examinations showed that in stages I and II there were no changes of oesophageal or gastric mucosae. In stage III, only deep ulcerations and stenosis were evident. Radiocinematography revealed essentially dysfunctions on apparently moderate lesions that were localised to the passage of Killian, the oesophagus, or the cardia.

Table 4  Healing of digestive tract burns

<table>
<thead>
<tr>
<th>Stage of lesions</th>
<th>No.</th>
<th>Healing time (days)</th>
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<tbody>
<tr>
<td>I</td>
<td>35</td>
<td>8 (oral feeding)</td>
</tr>
<tr>
<td>II</td>
<td>27</td>
<td>20-30 (TPN)</td>
</tr>
<tr>
<td>III</td>
<td>14</td>
<td>90 (TPN)</td>
</tr>
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</table>

examination and above all by endoscopy (Table 4). The 35 patients with stage I lesions who were maintained on oral nutrition healed completely in one week. In patients maintained by total parenteral nutrition according to the results of successive radiological and fibroscopic examinations healing without sequelae was obtained in 20 to 30 days in 27 with stage II lesions, and in 90 days in 14 patients with stage III lesions. The time taken for healing with total parenteral nutrition was invariably irrespective of the type of product ingested.

 Associated problems Mild epigastric tenderness on palpation persisted for one to two months. Rarely did it increase (one case) and was only rarely accompanied by guarding. Those presenting with haematemesis on admission showed resolution of this problem in less than a week in two cases and with considerable moderation in one patient in one other case. Inhalation bronchopneumonias were rapidly controlled with antibiotic therapy with the exception of one case who developed Mendelson's syndrome after inhalation of hydrochloric acid and who subsequently died.

Local complications Only in the 14 patients with stage III lesions were new complications seen during the course of their illness. Four oesophageal stenoses and one tight gastric stricture (Table 5) occurred in this group of patients. Other complications were extremely rare. One perforation situated posteriorly in the stomach occurred on the fifteenth day. There were no cases of new gastrointestinal haemorrhage or bronchopneumonias.

Surgical management All the patients with oesophageal strictures were treated with colonoplasty of the oesophagus—that is, four cases in all. In one case with a mid-gastric stenosis a simple resection followed by reanastomosis was possible. Normal oral nutrition was restarted 15 days after such an operation.

Table 5  Frequency of stenosis with total parenteral nutrition

<table>
<thead>
<tr>
<th>Stages of lesions</th>
<th>No.</th>
<th>Healing</th>
<th>Stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>35</td>
<td>35</td>
<td>—</td>
</tr>
<tr>
<td>II</td>
<td>27</td>
<td>27</td>
<td>—</td>
</tr>
<tr>
<td>III</td>
<td>14</td>
<td>9</td>
<td>5</td>
</tr>
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</table>
COMPLICATIONS RELATED TO METHOD OF TREATMENT

Complications of medical treatment

Endoscopy No problems were encountered during 94 endoscopic examinations undertaken a few hours after the ingestion of corrosive or during the course of 253 examinations undertaken either during the chronic period or during the course of follow-up.

Total parenteral nutrition Infections were the only problems to mark the clinical progress. Forty-one patients were infused for periods of 20 days up to four months, giving a total of 283 days of infusion. Infections occurred only in patients with stage III lesions. There were five cases of septicaemia, due to candida in four cases and to enterobacteria in one case.

Complications of surgical treatment

In the four patients who underwent colonoplasty operations only one case of postoperative dysphagia occurred, which improved with re-education after one month. In two cases haematomasis occurred and reoperation was necessary in one case. There were no examples of wound dehiscence at the plastic surgery reconstruction site.

OVERALL MORTALITY

All the four deaths occurred during the period of medical treatment and only in patients with stage III lesions involving the oesophagus and the stomach.

One patient died after 48 hours having had total destruction of the stomach after ingestion of hydrochloric acid and associated with shock and anuria.

Another patient died after 15 days with Mendelson’s syndrome, again after ingestion of hydrochloric acid. The third case, a man aged 65 years, died after three months from multiple pulmonary emboli and infection after ingestion of caustic soda. The fourth died after complete thrombosis of the superior vena cava caused by a polyvinyl catheter.

Discussion

ENDOSCOPY

With respect to corrosive lesions affecting the upper gastrointestinal tract most authors insist that early observation of the lesions is vital. This examination permits, firstly, confirmation of the presence of digestive tract lesions. The diagnosis of such lesions is, in effect, virtually impossible if one attempts to base this simply on the imprecise history obtained either from the patient or from the immediate family. On the other hand, functional and physical signs are either minimal or absent. Involvement of the otorhinolaryngeal area is inconstant and bears no clear relationship to lesions of the upper gastrointestinal tract, and other aspects—such as, for example, the nature of the ingestion act (self-poisoning or accidental), the nature of the product, the quantity of the product ingested, or the period of contact—are not very accurate indicators. Secondly, we have noted that the frequency of gastric involvement is an important prognostic factor in localising the lesions but that it is equally unpredictable from simple clinical examination or just oesophagoendoscopy. Thirdly, the examination enables us to appreciate the severity of the lesions and to adopt the appropriate therapeutic regimen. The classification of these burns into three degrees is based only on the extent and severity of the superficial lesions and might be improved upon by an appreciation of the depth of the involvement, perhaps with the help of radiocinematography.

The advantage of this method is that it separates serious and very severe burns which might become complicated and require treatment from benign ones, which for practical purposes may be ignored. In other words, this approach allows a rapid definition of the nature of the corrosive burns and allows one to make a prognosis. In addition, the inclusion of a fourth stage in this scheme to cover rare total destructive lesions of the upper gastrointestinal tract seems desirable. We believe with others that the fibroscope is to be preferred to the more rigid oesophagoscope because it is more flexible and because it allows the upper gastrointestinal tract to be completely assessed with far less trauma than hitherto supposed. In this connection, we have found it prudent to do an exploratory endoscopy as soon as possible after ingestion, so as not to cause haemorrhage or perforations of lesions that had become too advanced. In fact, from the evidence of successive endoscopic controls, it appears that the maximum intensive reaction in such lesions occurs very rapidly, probably before the end of the first hour, and thus this examination can be undertaken with no more risk at a fair interval after the initial incident. However, we continue to favour early endoscopic examination so that the more severe corrosion burns of the upper gastrointestinal tract are protected from unnecessary trauma.

In any event, in order not to increase trauma to the digestive tract mucosa, it is preferable not to practice endoscopy routinely except during the period of presumed healing. Radiological investigations are of little practical value in determining the state of the mucosa. On the other hand, it seems that it may be useful during the healing stage to localise an oesophageal stricture or to assess the condition of the stomach. However,
only endoscopy can verify directly the healing state of the mucosa and so indicate when surgical intervention can be performed without risk.

**TOTAL PARENTERAL NUTRITION**

This comprises the second component of our method. This approach has several advantages in cases of severe corrosive burns. Firstly, it protects the burn from trauma and infection, which may arise from oral nutrition or the use of nasogastric tubes, and diminishes the frequency of local mechanical complications and infections, which usually occur in 20%-30% of most series. The occurrence of inhalation bronchopneumonia is avoided in cases with deglutition problems. Mild lesions are not made worse. The frequency of stenoses developing is probably reduced. The results from the literature show a 4%11–27%18 incidence of strictures after corrosive ingestion treated by classical methods, and the figures exceed 43%13–50%9 if one considers only very severe lesions (but there are no precise figures on this point, because of the absence of similar initial and control endoscopic examinations). These figures are considerably higher than those obtained in patients treated only with total parenteral nutrition. It is possible that repeated trauma to the injured or necrotic mucosa may exacerbate the fibroblastic reaction that normally occurs and so gives rise to stricture formation14, a phenomenon which, theoretically, could be avoided or at least limited by total parenteral nutrition. Second, total parenteral nutrition also rapidly restores the stage of well-being and reverses the effects of the hypercatabolism associated with the acute phase. On this point it is difficult to suggest an optimal caloric intake but it appears to be around 40–50 kcal/kg/day, a level that leads to rapid healing and prevents the occurrence of perforation or haemorrhage. Secondary bacterial infection of digestive tract lesions arising from intravenous catheters has been a problem, while, on the other hand, dissemination of infection from an infected upper gastrointestinal tract lesion, which is from the start not inaccessible to normal antibiotic therapy, has been more frequent. Early total parenteral nutrition should enable the upper gastrointestinal tract to be isolated from the standpoint of infection.

**THE TERAPEUTIC NIHILISM**

This appears to be the method of choice at the moment. Results from the literature clearly show the effectiveness and the danger of using steroids to avoid the formation of strictures.15 Systemic use of steroids and antibiotics is not indicated and, in fact, is dangerous in the way used hitherto. Oesophageal dilatation by bouginage, which can cause further local trauma, does not seem indicated as a method of choice.17

**INDICATIONS FOR SURGERY DURING HEALING PHASE**

This is the optimal way of treating the sequelae to corrosive burns, and postoperative complications are exceptional and rapid correction easily achieved.18 Whenever possible and for maximal beneficial effects all reparative surgery should be avoided during the acute period when tissues are very necrotic and inflamed.19 This approach is, after all, much easier if total parenteral nutrition reduces considerably the frequency of local complications during the early phases of corrosive burns. Total destruction of the oesophagus and stomach with intraperitoneal contamination with the ingested product could be treated only by emergency oesophagogastrectomy followed by total parenteral nutrition and later reparative surgery.20 Acid inhalations causing bronchial erosions leading to haemoptysis can be dealt with only by membrane oxygenators. Enforced bedrest in the aged leads to complications which, in turn, may be an indication for jejunostomy feeding. Thrombosis of the superior vena cava could probably be avoided by the use of silastic catheters. It is difficult to compare the mortality rates obtained in our proposed method of treatment with the 10%21 and 26%1 obtained elsewhere, because of the difference in the management protocol in the series (Table 6). Only a prospective study could confirm our results but from an ethical standpoint and at this moment in our own experience such a trial would be difficult to conduct.

The therapeutic management that we have outlined allows an immediate prognosis to be made and an evaluation of the high risk group of patients in whom therapeutic efforts may prevent stenosis.

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