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Acute corrosive oesophagitis

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This paper concerns the diagnosis, pathology, and treatment of oesophageal injuries caused by swallowing acids or alkalis. The treatments recommended aim not only at saving life in the early phase but in preventing intractable stricture later on.

A review of the literature on the management of corrosive oesophagitis with subsequent stricture formation reveals a wide variation of opinion as to suitable treatment. European methods were at variance with those of North America. General practitioners, paediatricians, and internists tackled the problem from different points of view, and even among otolaryngologists there was considerable divergence of opinion. In the past, much of the treatment began after the formation of strictures, which inevitably involved prolonged bouginage with lengthy hospitalization on both an in-patient and out-patient basis. In recent years there have been radical changes in therapy, based on the concept of prevention of stricture formation.

Over a period of 10 years the authors have devised a plan of treatment based on the pathology of this condition and reported experimental investigations.

About 75% of cases of acute corrosive oesophagitis are caused by the ingestion of strong caustic solutions. Acids cause more superficial injuries in the oesophagus, while in the stomach they produce more serious damage, partly because they are not neutralized by gastric juice as alkalis tend to be. Many of our common lye preparations have the consistency and appearance of milk and thus are 'fair game for little hands'. Lye in the form of crystals is the most dangerous medium, producing, even in small amounts, intense, deep burns with all their sequelae.

PATHOLOGY

A better understanding of the pathology of acute corrosive oesophagitis, gained by direct visualization by oesophagoscopy and post-mortem studies, is the foundation of our present-day treatment.

Briefly, the course of corrosive lesions of the oesophagus may be divided into three phases:

1. ACUTE NECROTIC PHASE Death of cells occurs by coagulation of intracellular protein. Intense inflammatory reaction develops in the living tissue surrounding the overlying area of necrosis. There is thrombosis of the vessels and bacterial and haemorrhagic infiltration in the underlying layers. This phase occurs one to four days following injury.

2. ULCERATION GRANULATION PHASE When the superficial necrotic coagulated tissue sloughs at three to five days, an ulcerative, acutely inflamed base remains. During this phase early reparative processes are seen. By the end of the first week all layers of the oesophagus are involved in inflammatory oedema, with fresh granulation tissue appearing superficially. New blood vessels and fibroblasts are seen during this stage. The granulation tissue fills in the defect of sloughing areas, and collagenous connective tissue is seen developing at from 10 to 12 days. Significant repair response is first seen at five to seven days. Grossly, the oesophagus is weakest during this period.

3. CICATRIZATION AND STRICTURE FORMATION This occurs at the beginning of the third and fourth weeks. The collagenous connective tissue formation begins to contract, causing narrowing of the oesophagus. Adhesions between the granulating areas may occur, with resultant pockets and bands. Degeneration of both muscle and nerve tissue is seen. After the inflammatory reaction has subsided, healing is accomplished by replacement of the submucosa and muscularis coats with dense fibrous tissue layers. The maturation of the superficial inflammatory tissue progresses slowly over the first six weeks. The inflammatory reaction subsides following re-epithelialization of the oesophagus at four weeks to three months. This is the period when the greatest clinical vigilance must be maintained for signs of stricture.

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Because of the pathology and change, the physiological behaviour of the oesophagus may be permanently altered with a decrease in the peristaltic movement, and secretory activity.

The brunt of the corrosive injury is usually borne by the retrocardiac portion of the oesophagus, and here the most severe grade of stricture is common. Fatti, Marchand, and Crawshaw (1956) describe four grades of stricture:

Grade I The fibrosis is circumscribed, confined to less than the total circumference of the oesophagus, and to a short segment in length, the so-called 'shelf strictures' which never cause significant narrowing of the lumen.

Grade II There is a localized annular stricture of less than 0·5 in. in length. The fibrosis does not penetrate beyond the submucosa and consequent narrowing is never pronounced, therefore treatment is not usually a difficult problem.

Grade III There is a dense stricture of less than 0·5 in. in length. Fibrosis extends through the outer muscular coats and perhaps the lumen which is lined by epithelium. The lumen may be reduced to thread size.

Grade IV There is a tubular stricture with dense fibrotic walls, perioesophageal adhesions, and marked luminal narrowing. Because of the extensive destruction, epithelium cannot re-bridge the gap and the lumen is lined by fibrous and granulation tissue. Such strictures are always found to be difficult to treat.

DIAGNOSIS

In severe cases the patient experiences a burning sensation of the lips, the mouth, and pharynx with exquisite pain beneath the sternum, in the back, or in the epigastrium. He appears pale and anxious. Salivation is abundant. The pulse is thready and rapid. If vigorous treatment of the general condition is not started, a state of shock ensues that can end in coma and death. In the lighter forms, pain is more or less acute in the interscapular region or in the base of the neck or beneath the sternum. The lips, the gums, the internal surface of the cheeks, the tongue, and the pharynx are hyperaemic. The mucosa desquamates.

The presence of injury in the oropharynx cannot be considered a reliable criterion for the institution of therapy for oesophageal burns. In published series it is recorded that 25–50% of patients with oropharyngeal lye burns develop strictures. Also recorded are cases that develop stricture formation of the oesophagus without evidence of oropharyngeal burns.

The importance of demonstrating the site and extent of the burn by adequate early oesophagoscopic examination seems obvious. If there is no oesophageal lesion, dilatation is not indicated. Very early and aggressive dilatation may be hazardous if the lesions are extensive. Careful oesophagoscopy appears to be the best means of early accurate diagnosis, and only by this means can the physician plan his treatment on a logical and pathological basis. Early oesophagoscopy has two advantages:

1. The patient without oesophageal burns, which comprise up to 50% of cases, can avoid prolonged hospitalization.

2. The oesophagoscopy provides some knowledge of the extent and severity of the oesophageal damage. The limitations of oesophagoscopy must also be recognized and appear to be the following:

(*a*) It is difficult to evaluate the depth of any burn with absolute certainty by observing superficial epithelial necrosis.

(*b*) When a severe burn is encountered in the upper third of the oesophagus and the oesophagoscope is not passed beyond this area, the involvement of the middle and lower thirds is not known.

(*c*) Occasionally, and when directly visualized early, the oesophagus may be interpreted as normal with, nevertheless, later development of stricture.

It is impossible to decide from radiography alone what form of treatment is indicated. In the early stage the passage of opaque material through the oesophagus is slow but otherwise not very abnormal. The most pronounced changes are observed when stenosis is reached, long *after* the period when vigorous preventive treatment for stricture should have been instituted.

The therapy of acute corrosive oesophagitis has undergone radical changes in recent years, and current thinking is now centred on the prevention of stricture, rather than on the treatment of the well-established oesophageal narrowing.

The magnitude of oedema, thrombosis, and presence or absence of bacterial invasion are factors as important as the chemical injury in determining the fate of the muscularis and ultimate stricture. Control of the inflammation by antibiotics promotes and hastens repair of the injured oesophagus. Acute necrosis is shortened and the ulcerative granulation tissue and cicatrization phase occurs earlier.

Antibiotics are best continued until there is re-epithelialization at four to eight weeks, depending on the severity of the burn. Antibiotics will not prevent stricture formation but will help to promote optimal healing conditions. If steroids are given, antibiotics may be discontinued after one or two weeks.

Experimentally, there is an inhibition of both acute and chronic inflammatory reaction, granulation tissue formation, and subsequent stricture formation with steroid therapy. Likewise, steroid-treated animals do not show the thickened, indurated oesophageal musculature that controls develop. Experimentally, although there is a decrease in the incidence and degree of oesophageal strictures from alkali corrosives when steroids are used, it is evident that they can and do develop in spite of steroid therapy. The oesophageal stricture found in steroid-treated dogs is less firm and more pliable than that in control animals.

Steroids, ideally, should be continued until there is complete absence of all inflammatory reaction and fresh granulation tissue. This occurs when re-epithelialization is present at 1–3 months. Patients receiving steroid therapy must be followed by intermittent oesophageal observation, especially in the first two months, when 80% of clinical strictures appear.

Despite the fact that the dosage and length of treatment with steroids have varied in reported clinical series, the results have been encouraging. Over 100 cases of proved acute corrosive oesophagitis have been reported with only 11 strictures, or approximately 90% success.

Until recent years the management of corrosive oesophagitis and stricture was the prerogative of the endoscopist. In the acute stages prophylactic bouginage was recommended. This was done with varied frequency and, in the case of subsequent stricture, had usually to be continued for months or years—a prospect never eagerly anticipated by patients or physicians. Bouginage has a definite place in the treatment of corrosive oesophagitis, but the persistence of prolonged endoscopic techniques, apart from the accidents due to inexperience, have caused most of the fatalities in the past.

In view of these recorded observations and experimental studies we have pursued the following regime in the treatment of all corrosive burns of the oesophagus for the past 10 years.

All chemical burns of the oesophagus are inspected by means of oesophagoscopy under general anaesthesia as soon as possible after the injury, preferably in the first 48 hours.

If at oesophagoscopy there appears to be little or no involvement of the oesophagus, local treatment is administered to the oropharynx. If the child can swallow citrus juice in the case of a lye burn, it may be of value in the first two or three hours. Gastric juice will neutralize lye reaching the stomach and gastric lavage and the induction of emesis are contraindicated. Repeated swallowing of small amounts of olive oil is soothing, and a soft or liquid diet may be given.

If the endoscopist finds mucosal burns of the oesophagus which are circumferential and from his experience are probably second, third, or fourth degree burns, then all measures must be taken to prevent stricture formation. It is much better to err on the side of radical treatment, remembering the prolonged, dangerous, and difficult treatment of this major surgical problem.

In our opinion, therefore, the injured organ, like other injured organs, should be put at rest to minimize the results of inflammation as much as possible. This has been recommended by many physicians, *e.g.*, Hoag (1937) and Belinoff (1940), but surprisingly has not had many adherents. We have used it for the past 10 years to our satisfaction and consider it to be the best treatment to date, for reasons that will be stated.

A plastic tube of a calibre estimated at the time of oesophagoscopy is introduced into the stomach through a cervical oesophagostomy or pharyngostomy. It is important that the calibre should not be too large or too small. Bardex catheters are ideal for children. Cervical oesophagostomy provides the opportunity to increase the calibre during healing. The tube is well tolerated even by small children, providing easy feeding facilities. The tube is retained for up to three months, depending on the condition of the wall as judged by direct inspection.

This tube is removed at two-weekly intervals, the oesophagus is inspected directly, and the pathology noted. Often the oesophagus seems to dilate rather than constrict around the tube and a larger calibre can be inserted. The length of treatment by the indwelling tube can be judged by the disappearance of granulation tissue and the re-epithelialization phase.

Antibiotics are used in all cases for at least two weeks, by which time the inflammatory stage has largely disappeared. Steroid therapy is instituted from the beginning and continued in the appropriate dosage until the re-epithelialization stage has been reached. The average recommended duration of treatment with steroids for moderately severe burns is from six to eight weeks, for mild

burns one month, and for severe burns three months.

In the few cases that have residual stricture formation after the above treatment we institute visual dilatation at endoscopy at one- or two-weekly intervals, but rapidly institute self-bouginage with Hurst's mercury bougies at home until the situation is well controlled.

In very severe burns and particularly in patients who develop long tubular strictures with a densely fibrotic wall and marked luminal narrowing, prolonged conservative treatment will be futile and even dangerous. Replacement of the oesophagus by colon transplant, sub-sternally and extrapleurally, is the method of choice. We have used this in seven cases over the past four years. The results have been excellent except in two suicidal patients who required bouginage at home because of severe burns of the pharynx at the site of the anastomosis.

We feel that the above method, using a splint for an injured organ, with the addition of direct inspection at intervals, the use of antibiotics and steroids, and an adequate way of feeding, is ideal to prevent the complication of chemical burns of the oesophagus. In favour of this view we suggest the following arguments:

Intermittent dilatations cause fissuring of the stricture, either in the whole wall or only in the submucosa. The trauma and bleeding give rise to proliferation of the fibroblasts and of granulation tissue and, after a while, to renewed contraction. Gradual stretching of strictures produces less reaction and a more permanent result than sudden dilatation. This is the experience in dilating the urethra, ureter, rectum, and other passages of the body.

Any sinus through soft tissue tends to enlarge from the continuous presence of a tube which passes through it, *e.g.*, a tube in an empyema or gastrostomy. This type of dilatation is so gradual that the reaction of the tissue is minimal.

The impact of food as the result of swallowing is an important factor in producing and maintaining secondary inflammation and irritation which leads to final closure of the oesophagus. This is evidenced by the fact that when food is administered through a gastrostomy opening as a substitute for feeding by mouth, the oesophageal constriction will frequently re-open and again allow food to pass.

The only successful treatment of laryngeal strictures has been the use of core moulds, which is similar to the above recommended treatment of oesophageal stricture. Intermittent laryngeal dilatation has resulted largely in failure.

Our enthusiasm for this method was recently enhanced by results obtained in two children who were admitted with fully formed multiple lye strictures of the oesophagus, and almost complete dysphagia. They were subjected to our recommended treatment for six weeks. Subsequent oesophagograms revealed that one child had a minimal residual stricture in the upper third of the oesophagus, which resolved in six months without dilatation. The other had a normal oesophagogram on completion of treatment and it has remained so.

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