CORROSIVE STRICTURE OF THE OESOPHAGUS

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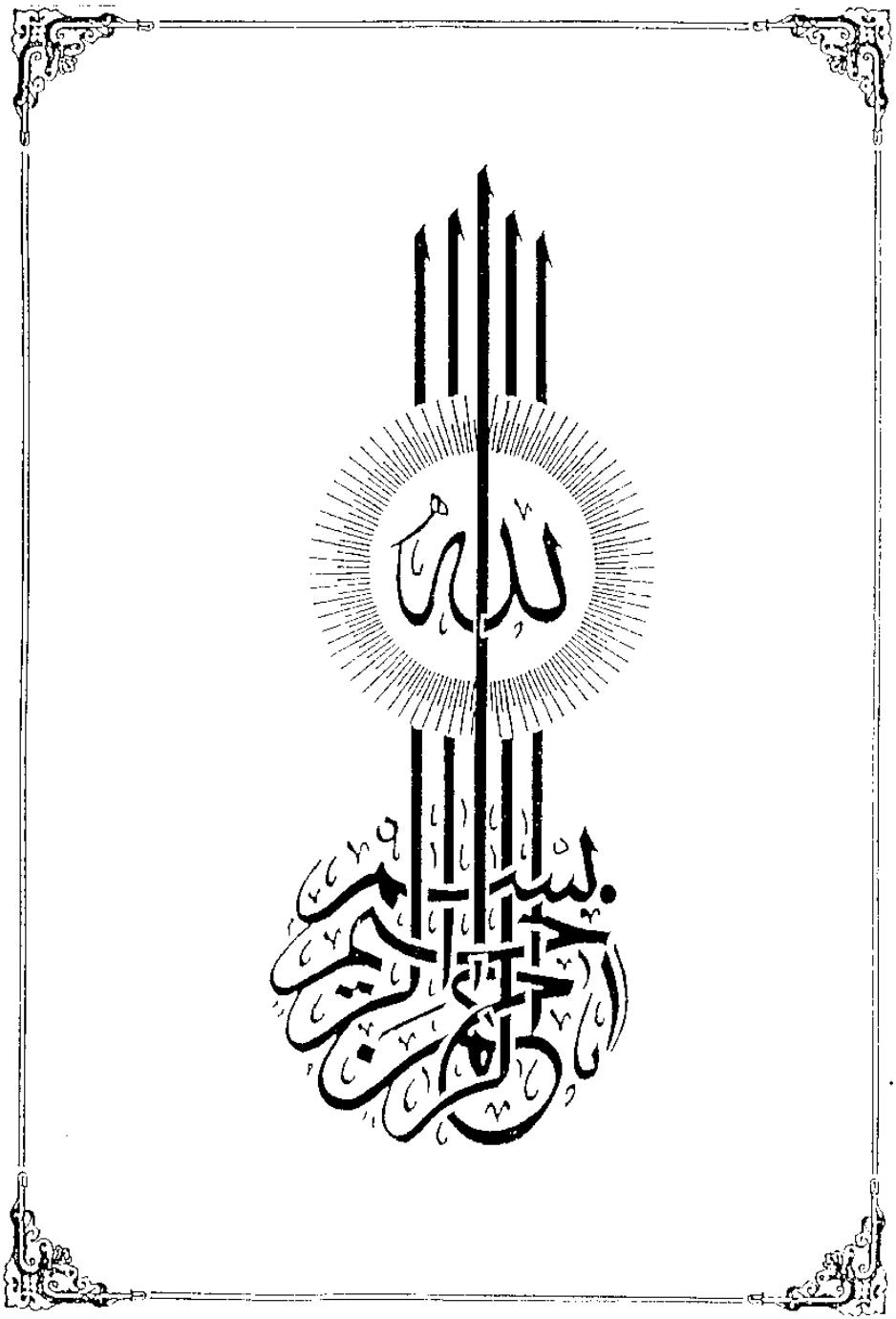

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Introduction

Corrosive injury to the oesophagus is a problem of considerable concern because, if not rapidly fatal, is often the precursor of a dense stricture. Caustic injury of the oesophagus is multifactorial and varies with the extent and depth of injury.

The enormous improvement in the outcome of corrosive injury is due chiefly to the introduction of antibiotics, cortisone and the development of oesophageal surgery.

Aim of the work

This essay addresses the endoscopic and clinical challenges encountered in corrosive injury to the oesophagus.

Early, accurate knowledge of the severity of the injury facilitates the administration of appropriate therapy.

Also, it is trying to conclude the role of each type of therapy to be able to choose the best method in each individual case.

Etiology

Caustic injury to the oesophagus follows the ingestion of a strong alkali or strong acid (Cello et al.,1980). About 80% of caustic injuries occur in children under 5 years of age who accidentally ingest strong household cleaners (Ferguson et al.,1989). In adults, it represents attempted suicide in psychiatrically disturbed patients (Borja et al.,1969).

The most common chemicals implicated in such corrosive burns of the oesophagus are alkaline caustics, acid corrosives and household bleaches . Alkaline caustics consist of sodium hydroxide (the active ingredient in household lye and drain cleaners), sodium bicarbonate (washing soda), sodium metasalicylate (dish washing detergent) and ammonia water (household cleaners) (Skinner et al., 1976).

Commercial lye is the causative agent in the majority of cases . Lye contains sodium hydroxide or potassium hydroxide in solid (100%) or liquid (10% to 30%) form as part of commercial drain cleaners or washing powders. The less injurious bleaches and household ammonia often contain ammonium hydroxide, sodium hypochloride, calcium hypochloride or hydrogen peroxide.

Lye tends to be odorless and tasteless, and therefore causes most pediatric caustic injuries (Middlekamp, 1969). Ingested lye in solid form may cause deeper injury but tends to adhere or stick to buccal, pharyngeal and proximal oesophageal mucosa, thereby precluding severe oesophageal or gastric injury (Borja et al., 1969). Liquid caustics

commonly produce much more extensive oesophageal and stomachic necrosis (Cello et al., 1980), and occasionally even trocheoesophageal and oesophagoaortic fistulas (Estrera A et al., 1984). If the patient survives the acute phase, a lengthy stricture often develops.

In contrast to lye , strong acids have an offensive odor, a bitter taste and hurt the lips, buccal mucosa and hypopharynx resulting in rapid expulsion if taken accidentally. The acid compounds usually seen in liquid toilet cleaners and anti-rust compounds contain hydrochloric acid. Battery acid includes sulfuric acid (Kirsh et al., 1976).

Ingestion of strong acid characteristically produces greatest injury to the stomach especially the antrum with the oesophagus remaining intact in over 80% of cases . The result may be immediate gastric necrosis or late antral stenosis (Dilawari J.B. et al., 1984).

Nearly all severe injuries are caused by strong alkali. Weak alkali and acids are associated with less extensive lesions (Postlethwait R.W., 1983).

In recent years , oesophageal burns have been reported with increasing frequency from ingestion of Clinitest tablets used to test the urine for sugar. These tablets contain a significant amount of anhydrous sodium hydroxide, which is strongly alkaline, and that is why they cause oesophageal ulceration and frequently stricture formation. (Symbas et al., 1983).

In the departement of surgery, Wayne State University, Detroit, Mich, from July 1980 till February 1989, 34 adult patients (20 men and 14 women) were admitted for ingestion of caustic materials. In 19 patients the ingestion was accidental mostly due to alcoholic affection and in 15 patients ingestion was a suicide attempt. Ingested agents included hydrochloric acid (HCl) in 4 patients, sulfuric acid (H_2SO_4) in one patient, a strong alkali such as sodium hydroxide in 15 patients, liquid bleach in 8 patients, a strong detergent solution in 4 patients and ammonia in 2 patients.

In Egypt, ingestion of caustic substances is commonly seen in lower socioeconomic class families (Ragheb et al., 1976). Commercial lye which is a solution of caustic soda or caustic potash is the most frequent agent implicated because of its faulty keeping in drinking bottles. Next in frequency are iodine, sulfuric acid, bleach,ammonia and drain cleaners.

Pathology

The exact extent of injury is directly proportional to the concentration of the chemical and the length of time the irritant remains in contact with the mucosa.

A strong alkali produces liquefaction necrosis which involves dissolution of protein and collagen, saponification of fats, dissolution of mucosal lipoproteins and deeper penetrating injuries. Acids produce a coagulation necrosis involving eschar formation which tends to shield the deeper tissues from injury. So acids usually do not result in as serious injury as lye ingestion.

Depending on the agent, the amount ingested, the concentration, and duration of exposure or contact, sloughing of the mucous membrane, edema and inflammation of the submucosa,infection, perforation and mediastinitis may develop.

All ingested corrosives may affect to a varying degree the oropharynx, larynx, oesophagus, stomach or even small intestine and colon. The resultant injury can be superficial or deep. The superficial burns may be limited to the bowel wall and are manifested by hyperemia, edema, blister formation or superficial ulcertion. Deep or third degree burns extend through the full thickness of the oesophageal wall and may even affect adjacent mediastinal tissues or penetrate into the pleural or peritoneal cavities.

In all instances, the burn wound goes through an acute, subacute and chronic (or cicatricial) phase. The acute inflammatory phase occur in the first few days following injury and is characterized by tissue destruction, inflammatory reaction, vascular thrombosis and secondary bacterial infection. During the subsequent subacute phase which may last as long as two weeks depending on the severity of the injury, all necrotic tissues are spontaneously debrided and granulation tissue is formed by fibroblastic and vascular proliferation. The injured oesophagus is potentially weakest during this intermediate phase (7-14 days post-injury). Symptoms of pain and dysphagia may well improve or even disappear during this peroid. The third or final phase is that of re-epithelization and scarring. The process of epithelization is usually complete by the sixth week following injury but maturation and contraction of the scarred oesophageal wall may continue for several months, during which progressive luminal narrowing may develop.

The severity of caustic injury can be categorized into 3 groups based on endoscopic examination:-

Grade I injury includs mucosal edema, erythema, hyperemia or superficial erosions that extend to the muscularis mucosa.

Grade II injuries are often exudative, with blisters or frank hemorrhage and extend through the submucosa into the underlying muscular layer.

Grade III injuries have extensive ulceration into the muscular layer , have eccentric hemorrhage with gray or black coagulative necrosis, show proximal luminal dilation with atonic muscles or have perforation with mediastinitis or peritonitis (Sugawa et al., 1981)

Gastric changes occur quickly after alkali ingestion, whereas, oesophageal changes in grade III injury often progress to stenosis over a peroid of 4 to 10 weeks.

Once a strong alkali is ingested, oesophageal dysmotility and reflux may prolong the oesophageal exposure time and increase tissue destruction. The resultant gastric injury parallels the ingested volume and the preingested gastric contents, which may dilute the alkali. Prolonged exposure causes severer necrosis, vascular thrombosis and bacterial overgrowth that aggravate the necrosis.

The usual sites of stenosis as demonstrated by endoscopy and esophagograms are at the levels of the bronchial crossing, the cricopharyngeus region and the diaphragm. At those sites reflex segmental spasm causing delay in the passage of corrosive material and prolonged contact with the esophageal wall. The greatest effect of the swallowed corrosive thus can be expected at one or more of these sites. So these are the sites of the esophagus with the most severe injury and the most frequent sites of stricture formation . Palmer (1963) stated that 20% of patients showed maximal injury in the upper one third of the esophagus, another 20% of patients showed maximal injury in the middle third of the esophagus, 30% showed maximal injury in the lower third and the remaining 30% showed widespread injuries. Postlethwait (1979) collected several series from the literature totalling 1682 cases with 36.9% of cases were having injuries in the upper third of the esophagus, 48.8% of cases having injuries in the middle third of the esophagus, 15.1% of cases having injuries in the lower third of the esophagus and 22% of cases were having multiple injuries of the esophagus.

The site of the esophageal injury was proved to be affected by the nature of the corrosive agent. Alkaline caustics do less harm in the lower part of the esophagus and rarely injure the stomach because much of the swallowed material is regurgitated and neutralized by the acid vomitus which was mixed with gastric juice. Ingestion of acids has more dangerous effects in the lower third of the esophagus as the action is potentiated by the acidity of the vomitus, (Ragheb et al., 1976).

Clinical Features

Symptoms:

Systemic symptoms roughly parallel the severity of the caustic burn (Symbas P.N. et al., 1983).

After ingestion of the material, moderate to severe pain begins in the lips, mouth and pharynx and then spreads to the neck and later to the chest. If only a slight burn has been sustained, moderate soreness with swallowing may remain for a peroid of several days with few or no other symptoms. After several days healing is complete and no further difficulty ensues (Marchand, 1955).

With a burn of moderate degree, the pain persists and food and at times liquid cannot be taken. Burns of mouth and pharynx will usually result in prevention of ingestion of any material. The edema and spasm in the oesophagus further contribute to dysphagia (Viscony, 1971). The affected oesophagus almost invariably becomes infected with resulting fever and tachycardia, which may be accentuated by the pulmonary involvement. Improvement is usually evident by the end of first week in moderately severe burns. The oral burns will have improved, so that pain is less severe. In the oesophagus, the acute edema will have subsided and the necrotic tissue sloughed. The febrile reaction subsides, (Cleveland, 1973).

Patients with severe oesophageal burns rapidly become critically ill. All layers of the oesophagus are usually involved in these cases, including the perioesophageal tissues. Patients often experience chest pain, dysphagia, vomiting (which may be severe) and drooling of large