COMPARATIVE STUDY BETWEEN COLON BY PASS AND INTERPOSITION FOR POST CORROSIVE ESPOHAGEAL STRICTURE

THESIS

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By

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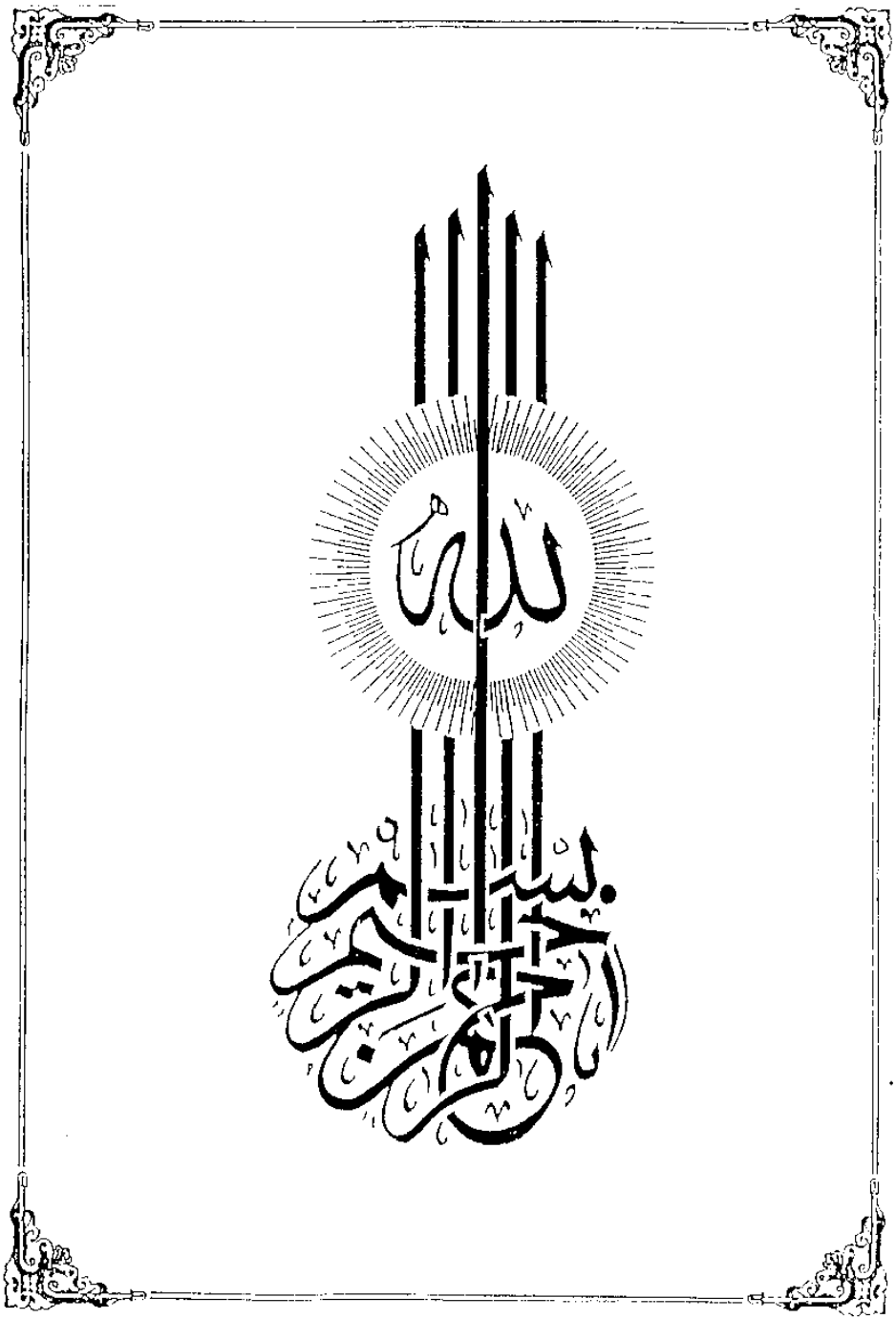

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CORROSIVE ESOPHAGITIS IN CHILDREN

Introduction

Corrosive injuries of the esophagus occur predominantly as a result of accidental swallowing of caustic agents in young children, and attempted suicide in adolescents *(Cello J.P. et al., l980).* Eighty percent of caustic injuries occur in children 5 years of age or younger *(Hawkins D.B.et al., l980).*

Serious consequences of ingestion are devastating when they occur requiring accurate and aggressive treatment. Caustic ingestion seldom results in systemic derangement. Inhalation injury, mediastinitis, gastritis and peritonitis are rare and tend to occur in younger children *(Ferguson M.K.et al., l989).*

Esophageal stricture is the most common sequalae of corrosive injury, its prevention by initial treatment and its successful management when established are the hall marks of patient care.

The most common chemicals implicated in such corrosive burns of the esophagus are alkaline caustics, acid corrosives and houshold bleaches.

Alkaline caustics consist of sodium hydroxide (The active ingredient in houshold lye and drain cleaners), sodium Bicarbonate (washing soda), sodium metasalicylate (dish washing detergent) and ammonia water (household cleaners) *(Skinner D.B. et al., l976).*

In Egypt, ingestion of caustic substances is commonly seen in poor socioeconomic class families *(Ragheb N.J.et al., l976).*

Commercial lye which is a solution of caustic soda or caustic potash is the most frequent agent implicated *(Bahnassy A.F.et al., l989).*

Potash is easily available in the Egyptian black market, it has an attractive appearance in cystalline or liquid forms, faulty kept in drinking bottles and cups and easily mistaken for sugar candy or milk *(Loutfi A.H. et al., l989).*

Detergents and Bleach virtually cause only mild esophageal irritation, which heals without adverse sequalae. Acids and alkalis, however, are capable of producing dangerous injuries ranging from chronic esophageal and gastric strictures to acute multiorgan necrosis and perforations.

Alkalies are the most destructive, causing liquefactive necrosis that facilitates deep penetration, whereas acids typically produce coagulative necrosis that tend to limit, to some extent; the depth of injury *(Haller J.A., 197l).*

Tucker J.A.et al., 1979, have summarized concisely the history of treatment of caustic ingestion.

Until 1900, treatment of esophageal stricture was limited to blind esophageal dilatation or in extreme cases to the placement of feeding gastrostomies.

The introduction of hardened rubber dilators in the mid 19th century provided the first reliable technique for dilatation of esophageal strictures.

With the development of a lighted esophagoscope by *Chevalier Jackson, in 1902,* direct visualization and direct dilatations of strictures became feasible. A significant improvements in dilatation techniques was provided with the introduction of retrograde dilatation through gastrostomies by *Gabril tucker* in the 1920s .

During the past 50 years, medical advances had improved acute care of corrosive esophageal injuries. Antibiotics introduced in the l940s, have reduced the incidence of stricture formation by reducing infection rate in the injuried esophagus. In the fifties steroids became a major centributor in the management of acute corrosive injuries. Hoping by its effect on the inflammatory process and the fibroblastic activity, it would decrease the incidence of chronic stricture formation following corrosive injuries *(Adam JS, l982).*

Many debates were against the use of steroids, and the value of its use was clearly evaluated by *Andreson K.D. et al., l990* who had shown in a randomized prospective study that the use of corticosteroids did not reduce esophageal stricture formation after caustic ingestion stricture formation was related only to the severity of injury *(Anderson K.D., l990).*

Pathophysiology:

The nature and extent of esophageal injury following caustic ingestion are determined by; type of agent (Acid or Alkali); state of agent (solid or liquid), concentration and amount of ingested agent and the length of time the irritant remains in contact with the mucosa *(Ashcraft K.W. l974).*

The different mode of action of alkalies and acids account for their differing potential for clinicaly significant esophageal corrosion.

Corrosive alkalies, including potassium and sodium hydroxide, produce the most severe esophageal injury by penetrating tissues, saponifying lipids, dehydration of cells and dissolving and coagulating proteins and collagen.

The result is brown, soft, friable eschar, (liquefactive necrosis), beneth which destruction continues until the alkali is neutralized.

Acids produce hard eschars either by their action as reducing agents (hydrochloric acid) with conversion of esophageal proteins to salts of the acids or by dissecation (sulfuric acid) with destruction of carbon containing tissues. The contact reaction minimizes continuing penetration and reduces ongoing esophageal injury as compared with the effect of alkali *(Jelenko C, l974).*

Ingested acids tend to cause significant gastric damdge with relative sparing of the esophagus, although severe esophageal injury can occurr *(Goldman L.P., l984).*

The Reflex pyloric spasm occurring in response of contact with corrosive agent cause poaling of both acids and alkalies in gastric antrum resulting in severe damadge that can lead to antral stenosis or an hour glass type deformity. Experimental studies in dogs have shown that both cricopharyngeal and pyloric spasm occur after concentrated lye instillation into the esophagus. Contractions of the esophagus and stomach then propel the contents back and forth between the two organs for several minutes until gastric and esophageal atony occur *(Ritter, F.N., l968).*

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 effect in the lower third of esophagus as the action is potentiated by the acidity of the vomitus *(Ragheb M.I., et al., l976).*

Clinical manifestations:

Ingestion of corrosive substances in childhood is seldom witnessed, and is verified on the basis of a history of possible ingestion and medical assessment of the patient, every effort should be made to document the possible agent, its physical and chemical characteristics and its volume, it should be noted that not all patients with oropharyngeal lesions have esophageal injury *(Ganderault P. et al., l983).*

Conversely, the absence of oropharyngeal burns does not eliminate the need for esophageal visualization and evaluation. 3-5% of patients with endoscopically determined esophageal injury have no oropharyngeal burn *(Adam JS et al., l982).*

The natural progression of the corrosive affection to the esophagus follows regular lines; there is immediate oedema, eschar within about 48 hrs and mucosal sloughs begins. In 5 days fibroplastic activities takes place. Scars follow fibroplasia and then regenration of mucosa.

If the scar is circumferential in an area of small lumen, a stricture will form. These strictures mature regularly in l4 to 2l days. They will progress to total dysphagia, impeding the ability

to swallow even saliva in 2l days almost by clock *(Davis, M.V., l982).*

Clinical manifestations vary slightly according to the amount and character of caustic agent ingested *(Goldman LP et al., l984).*

Mild burns of the pharynx, esophagus and stomach may be asymptomatic.

Solid alkali usually causes burns of the mouth, pharynx and upper esophagus. The severe pain caused by these burns usually induces immediate expectoration so that little is swallowed, such burns cause excessive salivation as well. Examination of the mouth and oropharynx reveals areas of mucosa that are replaced by white to grey pseudo-membranes. Hoarsness, stridor, aphonia and dyspnea suggest the presence of laryngo tracheal oedema or actual destruction.

Liquid alkali is usually swallowed rapidly, causing less injury to the mouth and pharynx but extensive damdge to the esophagus, stomach or both. Dysphagia, odynophagia and aspiration may be present. Severe substernal pain, back pain, abdominal pain and peritoneal signs suggest that mediastinitis or peritonitis result from esophageal or gastric perforation.

With acids ingestion injury to the stomach is more common and therefore symptoms and signs are more localized to the abdomen.

Severe caustic injuries that result in esophageal or gastric perforation are associated with progressively severe septic and hypovalaemic shock until appropriate thearpy is instituted. When perforation does not occur, the acute clinical manifestations usually resolves within several days. Resolution is followed by a period of clinical improvement usually lasting for several weeks. Symptoms due to stricture formation in the esophagus or the stomach then become manifest. Strictures occur in only 10-25% of patients after ingestion of solid alkali *(Estera A. et al., 1986).*

However most patients who ingested liquid alkali have severe esophageal and sometimes gastric damdge. Acid ingestion is more likely to result in stricture or contracture of the antrum or pylorus.

Immediate diagnostic and therapeutic measures:

Patients with acute caustic ingestion should be admitted to the hospital. Early management should be directed toward stabilizing the patient and assessing the severity of lesion.

Vomitting should not be induced because of the nearly instantenous nature of caustic injury.

Attempts to dilute the agent by drinking water are of little value, in fact this may increase the problem by causing increased gastric distention or vomitting oral intake is with-hold and hypovolaemia is treated with appropriate intravenous infusion. Patients are carefully observed for signs of airway obstruction. Endotracheal intubation or tracheostomy may be necessary because of laryngeal oedema in severe cases . Antibioties should be started as soon as the diagnosis of significant esophageal injury is established to decrease the risk of pulmonary infection from aspiration and bacterial invasion through the damdged esophageal wall *(Moore W.R. l986).*

Steroids have been advocated in the aucte phase of caustic ingestion, to prevent stricture formation *(Haller J.A. et al., l97l).* However their efficacy has never been established, and their use may obsecure signs of sepsis and visceral perforations and impair healing. The use of steroids for caustic injury can not at present be recommended *(Oakes, D.D. et al., l982).*

For evaluation of the patient with caustic injury, cervical or mediastinal soft tissue abnormalities on intial plain films