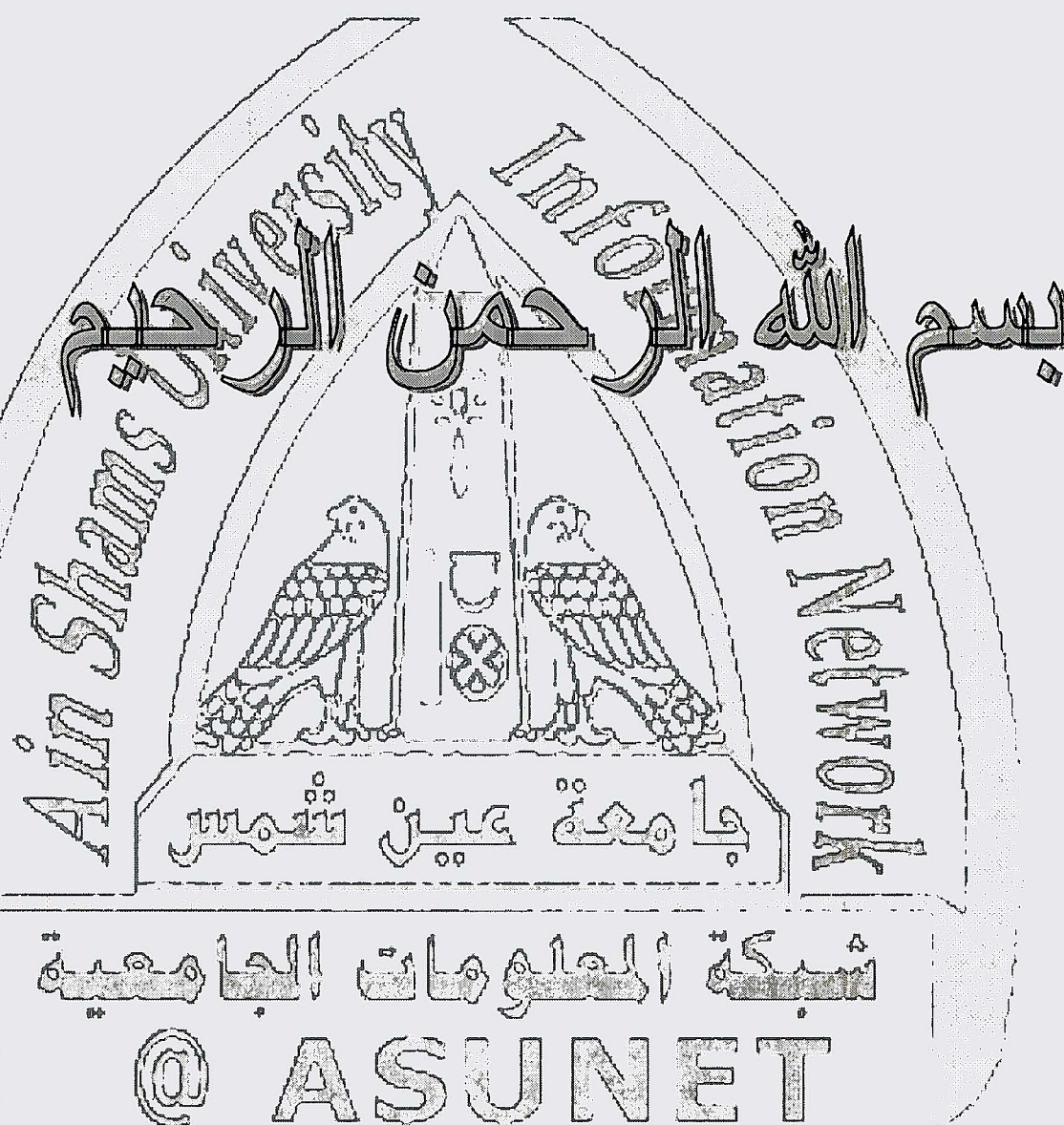




شبكة المعلومات الجامعية





شبكة المعلومات الجامعية

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شبكة المعلومات الجامعية التوثيق الالكتروني والميكروفيلم

بعض الوثائق الأصلية تالفة

Study On Esophageal Replacement by Colon : Interposition Through A Thoracic Approach Versus Transhiatal Approach In Benign Esophageal Lesions In Children

Thesis

Submitted in partial fulfillment of the requirement of the doctorate degree of general surgery

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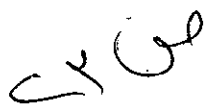
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Introduction

The question whether transhiatal (TH) or transthoracic (TT) resection is most suited for esophagectomy remains unresolved. Both approaches have their supporters, and there are several reports suggesting that TH approach is better in many aspects than TT approach, as well as other reports denoting the reverse. Advocates of TH resection are very enthusiastic about this approach; many of them recommend it for almost all patients in need for esophagectomy. They stress on the advantage of not making a chest incision, with decreased respiratory complications, the shorter operation time, the benefit of placing the upper esophageal anastomosis in the cervical region where leak, should it happen, is less dangerous and can be managed more easily with a better outcome than a thoracic leak.

Supporters of TT resection claim that TH is blunt, blind and could result in significant bleeding, chylothorax, airway injury and recurrent nerve paresis. They also stress on the fact that the given advantages of TH are either unproven, or achieved in the TT approach.

Huge number of studies were done to evaluate each approach, discussing its advantages and disadvantages; however, in few series, the authors did extensive review of their experience with both approaches and compared them together as regard to the operative complications and outcome of surgery.

In this prospective study, we compared both approaches for esophagectomy for benign lesions in children.

Esophageal Replacement

The esophagus has two functions: it propels ingested material by coordinated peristalsis induced by the swallowing mechanism, and at the same time, it inhibits reflux of the swallowed contents back. Appreciation of these important functions becomes readily apparent when abnormal esophageal motility patterns occur. This is particularly evident in infants with dysmotility disorders associated with esophageal atresia or in patients with chhalasia or achalasia and in those who have required a replacement of the diseased esophagus. With rare exception, a poorly functioning esophagus is usually better than any esophageal substitutes, and a decision to replace the esophagus must be considered very carefully.

A variety of complex and refractory problems are necessitating this procedure for occasional infants and children. The major indications for esophageal replacement include the following:

I- Stricture due to corrosive injury

Currently, it is the primary indication for total or near total esophageal replacement in children. Ingestion of a caustic substance is, unfortunately, still a major problem despite seemingly stringent laws of labeling and repeated warnings by the medical professionals. Due to the widespread use of lye by certain classes of people as an ingredient in homemade soap or as a cleaning agent per se, lye is easily available to children. Lye has an attractive

appearance, either in liquid or crystalline form, and it is easily mistaken for milk or candy ^{2,3}.

The exact frequency of chemical injuries cannot be determined, although Leape and his associates state that accidental ingestion of lye occurs in approximately 5000 children in the under five years of age each year united States ⁴. In Egypt, corrosive esophagitis is a problem of concern, it occurs most commonly in children about the age of five years. The patients are usually from lower class families ⁵. In the Netherland, an estimated 1,000 people per year are admitted to the hospital for caustic ingestion; the majority of them are children ⁶. The majority of chemical injuries are due to accidental ingestion by children. In adults, the chemical is usually swallowed with suicidal intent ⁷. The relative incidence of suicidal to accidental ingestion of corrosive fluids varies geographically: in Eastern Europe, the majority of cases are suicidal, in the Middle East, where corrosive materials, are widely used for domestic washing purposes, the injury usually occurs accidentally, certainly in children ⁸.

Etiology:

Alkali: Lye remains the most common caustic agent ingested, accounting for the majority of esophageal burns in series from Finland, the Soviet Union, Denmark, and the United States ⁷. Prior to 1967 in the USA, lye (sodium hydroxide or potassium hydroxide) was available primarily in particle or granular form ⁹, ingestion of this form of alkali causes such pain that the patient expectorates the caustic material and takes no more, thus limiting the injury to the oropharynx ⁹⁻¹³. In 1967, liquid forms of lye were

introduced by the household cleaning agent industry⁹. From 1967 to 1969, approximately 20 per cent of all caustic ingestion reports occurred with Liquid-plumber, a colorless solution of 30 per cent NaOH that was associated with a very high rate of esophageal injury and subsequent perforation. Because of the markedly increased toxicity with these concentrated liquid lye solutions, many have since been reformulated. For instance, Liquid plumber is now 5 per cent KOH intermixed with smaller concentrations of NaOH and sodium hypochlorite¹³. In Egypt, caustic potash is the classic injurious agent met with. Other forms of sodium or potassium hydroxide that have been associated with liquid caustic ingestion include oven cleaners and some liquid cleanser products.

Another cause of alkali induced esophageal injuries is disc battery ingestion. These batteries, which may contain manganese dioxide, silver oxide, or mercuric oxide, all contain an alkaline electrolyte that is usually a 45 per cent solution of KOH¹⁴. They may produce esophageal damage by one or all of three mechanisms: (1) pressure necrosis, (2) alkali or chemical injury from extravasation of the electrolytic agent, and (3) electrical current from a battery that is not exhausted¹⁵. A third cause is ammonia. Its alkalinity is related to formation of ammonium hydroxide with hydration, but it does not have the deeply penetrating characteristic of either sodium or potassium hydroxide¹⁴.

Acids: strong acids (pH <5) in the form of sulfuric or hydrochloric acid, are the second most common cause of caustic ingestion. Weak acids may be found in some household cleaners. These agents usually formulated as a 5 to 6 per cent solution, have a pH of approximately 5 and require either a

significantly higher amount or a longer contact time to act as significant caustics ¹⁴.

Medications: Kikendall and associates ^{16,17}, and other authors ^{18,19} reported a large number of medications to act as local caustics. These include various tetracyclines, potassium chloride in tablet form, ascorbic acid, chloral hydrate, and most nonsteroidal anti-inflammatory agents, as well as others.

Scierosants: local injection is associated with acute inflammation, ulceration and bleeding, submucosal fibrosis, and possible stricture formation. These changes can be similar to the pathology seen with true caustic ingestion ^{20,21}.

Pathology

The extent and severity of injury results from three factors: (1) the concentration of caustic, (2) the quantity ingested, and, (3) the duration of contact ⁽²²⁾. Alkalies dissolve tissue and, therefore, penetrate more deeply, while acids cause a coagulative necrosis, which limits their penetration. ⁽²³⁾

Alkali: The lesions caused by lye injury occur in *three phases*. **First** is the acute necrotic phase, lasting 1 to 4 days after injury. During this period, coagulation of intracellular protein results in cell necrosis, and the living tissue surrounding the area of necrosis develops an intense inflammatory reaction. **Second** is the ulceration and granulation phase, starting 3 to 5 days after injury. During this period the superficial necrotic tissue sloughs, leaving an ulcerated, acutely inflamed base and granulation tissue fills the defect left

by the sloughed mucosa. This phase lasts 10 to 12 days, and it is during this period that the esophagus is the weakest. **Third** is the phase of cicatrization and scarring, which begins the third week following injury. During this period, the previously formed connective tissue begins to contract, resulting in narrowing of the esophagus. Adhesions between granulating areas occur, resulting in pockets and bands. It is during this period that efforts must be made to reduce stricture formation ²³.

The location and degree of esophagogastric damage depend on both the concentration and formulation of ingested alkali. Krey ²⁴ demonstrated experimentally that a normal solution of sodium hydroxide (3.8%) if comes in contact with the esophageal mucosa for 10 seconds produces necrosis of the mucosa, submucosa, and longitudinal muscle fibers. The area of necrosis penetrated into the outer longitudinal muscle when a 10.7% solution was used. When a solution of 22.5% sodium hydroxide came in contact with the mucosa for the same length of time, necrosis of the entire esophageal wall with extension to the peri-esophageal tissues occurred. In their experimental work, Ashcraft and Padula ²⁵ demonstrated the hazardous nature of even diluted caustics on tissues.

Granular lye is slow to go into solution, so if only a small amount is ingested, the burns may be localized and not severe. In addition, the burning sensation in the mouth when granular lye is ingested often leads to spitting out the corrosive without much being swallowed ²². As a result, where 10-25% of patients develop esophageal stricture after granular lye ingestion, most patients with a history of liquid caustic ingestion are found to have a serious

(near- circumferential) esophageal burn, and almost all progress to extensive stricture formation ^{9,12}.

Acids: Strong acids cause coagulative necrosis covered by a protective eschar, similar to that seen with thermal burns ¹⁴. Weak acids with a pH >5, particularly the hypochloric acid that is present in bleach, are considerably less toxic. The esophagus is not much harmed because its mucosa is alkaline and the caustic has too brief exposure time during swallowing to damage the esophagus ²⁶.

Clinical Features:

Symptoms:

After the ingestion of the material, moderate to severe pain begins in the lips, mouth, and pharynx, then in the neck, and later the chest. Many times, vomiting and excess salivation follow ingestion of the lye. The subsequent symptoms depend on the extent and degree of the burn. If only a *slight burn* has been sustained, moderate soreness with swallowing may remain for a period of several days with few or no other symptoms. The child may voluntarily limit his or her intake to liquids, but after several days healing is complete and no further difficulty ensues.

With a burn of *moderate degree*, the pain persists and food and some times liquids cannot be taken. Burns of the mouth and pharynx will usually prevent the ingestion of food and liquids. The edema and the spasm in the esophagus contribute further to dysphagia. Respiratory symptoms may

develop rapidly or only after a period of several days. If the glottis or the major air passages are burned because of aspiration of lye, edema may rapidly bring about respiratory difficulty with cough and severe respiratory distress. If the respiratory passage injury is less severe or is caused by aspiration of oral and pharyngeal secretions, symptoms will develop more gradually and usually be less severe. The injured esophagus almost invariably becomes infected, with resulting fever and tachycardia, which may be accentuated by the pulmonary involvement. In the neglected patient, dehydration and weight loss become evident after a few days.

Improvement is usually evident by the end of the first week with the moderately severe burns. The oral burns will have improved, so that pain is less severe and the child is less reluctant to take nourishment. In the esophagus, the acute edema will have subsided and the necrotic tissue sloughed. The patient may actually be able to return to a full diet with the more superficial type of moderately severe burn. Even with the deeper burns, soft food may be taken without difficulty. The respiratory symptoms will improve, the febrile reaction subsides, and the dehydration is improved with the return to an oral intake.

In the *severe burn*, the patient rapidly becomes critically ill. All layers of the esophagus are usually involved in these cases, including the periesophageal tissues. Salivation and vomiting are severe and the pain is continuous. Respiratory difficulties may be prominent. The temperature and pulse rapidly rise and shock may develop. Mediastinitis, empyema, or tracheoesophageal fistula may bring about the death of the patient^{8,14,27}.