

# Caustic esophageal injury .. Some Controversies

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June/10/09

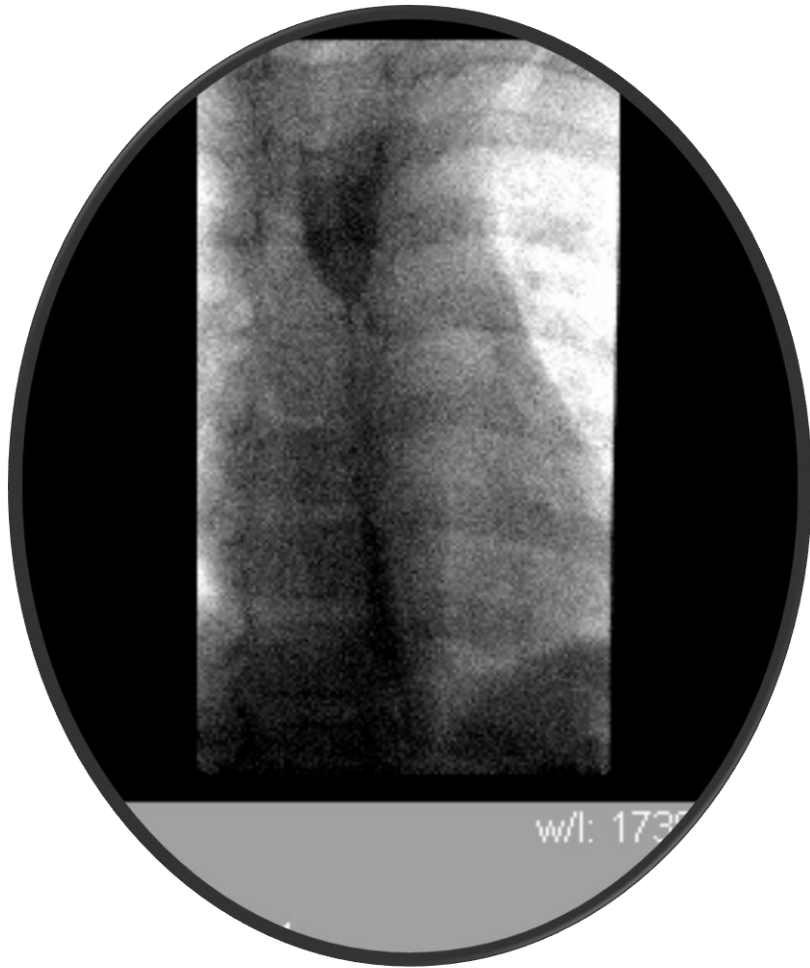
# The case

- 6 yrs, previously healthy
- Hx of Alkali caustic ingestion on Jan/07 → sever esophageal injury
- Subsequently, this complicated by esophageal strictures that require serial dilatations & esophageal stent

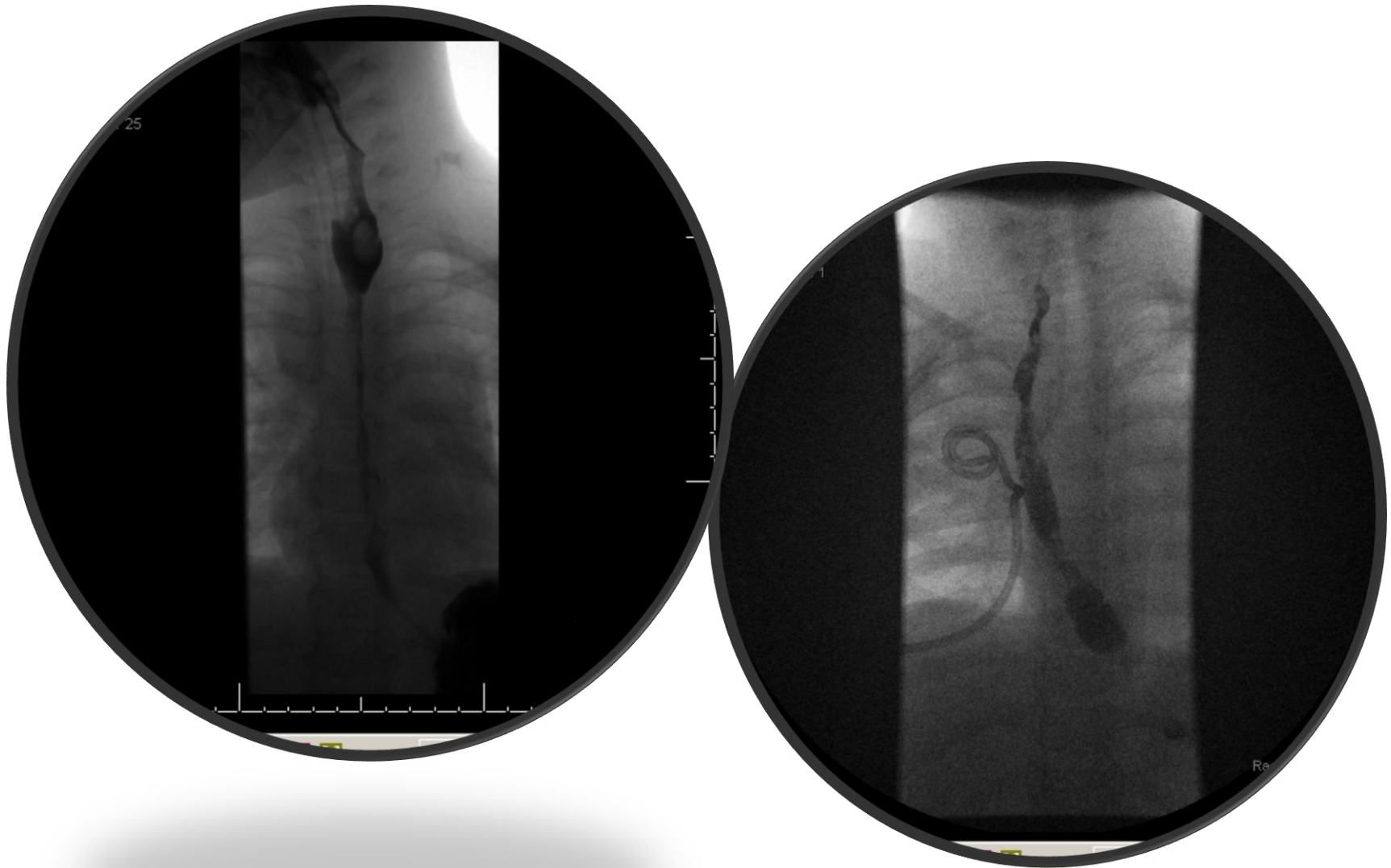
1 week after the ingestion



2 weeks after the ingestion



# Esophageal dilatation by intervention radiology

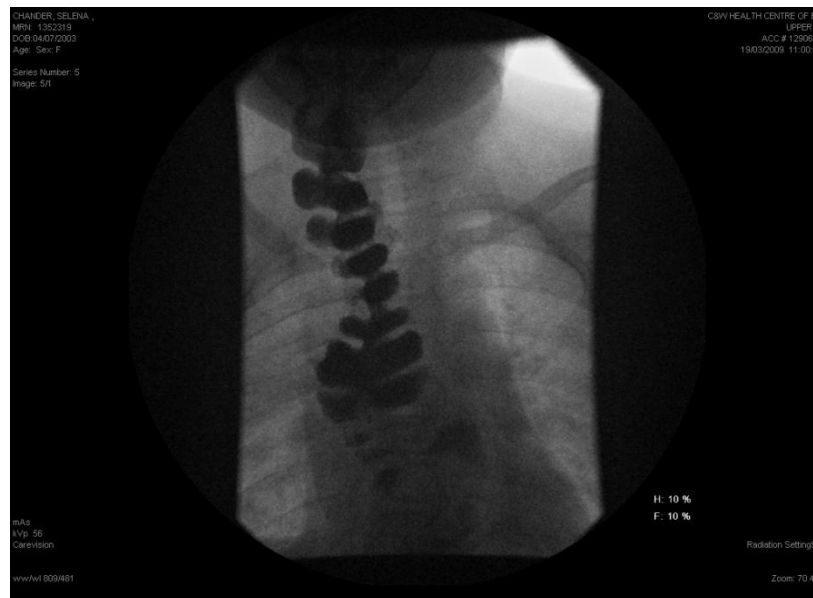


# The case

- Unfortunately, she ended by having esophagectomy with gastric tube replacement & GJ tube for feeding ( 1.4 yrs post ingestion)
- Postoperatively, the course complicated by mediastinitis, entero-pleural & entero-bronchial fistulae, aspiration pneumonia, left VC paralysis

# The case

- She ended up by removing the gastric tube connection & having colonic interposition (2.5 yrs post the ingestion)
- The course complicated by colic-colic leak & wound infection which required colostomy



# The case

- Significant retching.
- ? Would motility agent help her?



# Objectives

- **Quick overview of caustic ingestion epidemiology**
- **Discuss some controversial points:**
  - 1- using neutralizing/ dilution agents during the acute mx.
  - 2- When to do diagnostic EGD?
  - 3- The role of steroid?
  - 4- Is there a role for motility agents after surgical interventions?
- **Overview for the surgical mx for esophageal strictures**
- **The risk of esophageal Ca & its screening**

# Introduction

- Caustic ingestion is seen most often in **young children** between 1-3 years of age, with **boys** accounting for 50 to 62 % of cases
- Alkaline agents ( mostly household bleaches ) were more commonly ingested than were acids
- Other products most often involved included automatic dishwasher agents, laundry detergents, swimming pool products, and toilet bowl and oven cleaners.
- Esophageal burns have been reported in 18 to 46 % of caustic ingestions occurring in children

# Introduction

- Esophageal burns are rare with household bleaches (sodium hypochlorite) because they have a relatively neutral pH  
→ if occur mild & do not require treatment
- Industrial strength bleaches may be much more corrosive because of a higher concentration of sodium hypochlorite
- Solid caustic materials can produce deep burns of the oral cavity and esophagus ( adhere to the mucosa), but they are less likely to reach the stomach.

# Alkali vs Acids : mechanism of injury

- **Alkalis (pH >7) → cause liquefaction necrosis**  
→ early disintegration of the mucosa, allowing deep penetration and even perforation
- Agents with a **pH of 11 or higher** → more **severe burns**, (even with ingestion of small amounts)
- 2 factors affecting the chance & the degree of penetration:  
the concentration and the length of time the agent remains in contact with the mucosa

# Alkali vs Acids : mechanism of injury

- ***Acids (pH < 7)*** → cause ***coagulation necrosis***.
- The coagulum that forms on the mucosal surface may limit deeper penetration of the caustic substance
- The alkaline pH and squamous epithelium of the esophagus also help limit the severity of esophageal injury from acids.
- Esophageal injury seen in 6-20 % of acid ingestion
- Perforation of the esophagus is less common with ingestion of the alkali

# Stages of the caustic injury

- **ACUTE: Over the 1<sup>st</sup> week** → Following the initial necrosis (whether caused by alkali or acid), additional destruction caused by inflammation and vascular thrombosis will take place
- **SUBACUTE: By 10 days** → formation of granulation tissue and weakening of the esophageal wall ( esophagus is vulnerable to perforation → not a good time for EGD)
- **CHRONIC: By 3 weeks** → fibrogenesis and stricture formation ( perforation is less likely)

# Clinical manifestation

- Drooling, retrosternal or abdominal pain, hematemesis..
- Upper airway injury (stridor, hoarseness, nasal flaring, and retractions)
- Upper AW symptoms/signs are delayed by one- to two-hour delay with ingestion of powdered agents
- Deeper injury → esophageal perforation → mediastinitis, peritonitis, respiratory distress & shock.

# **NOT PREDICTORS**

- **Early** signs and/or symptoms may **not correlate** with the severity and extent of tissue injury.
- The presence or absence of any of symptoms or signs of corrosive ingestion does **not predict** the presence/absence or ***severity*** of esophageal or gastric burns.
- The presence or absence of **oral lesions also is a poor predictor** of esophageal injury.



# Clinical manifestation

- Dysphagia can be seen after the acute injury even without severe esophageal injury 2ndry to loss of motility & delayed transit time
- Prolonged esophageal transit time as measured by radionuclide testing may persist for several weeks following severe esophageal burns.

Cucchiara, S, C, et al. J Pediatr 1986; 108:907.

# Diagnostic evaluation

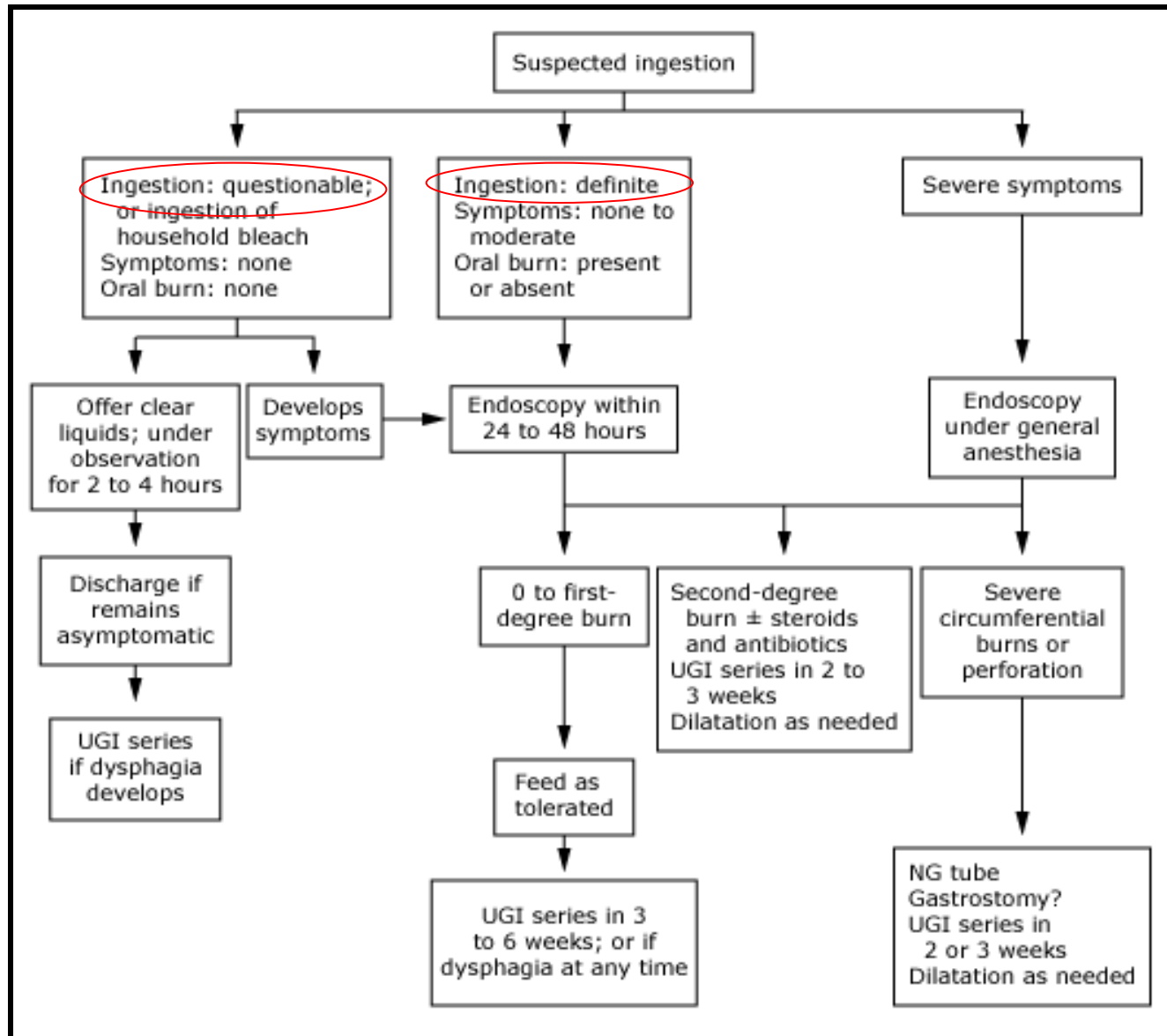
- **EGD :**
- Essential for determination of the presence/absence of the injury & extent → predict the prognosis
- Between **24-48 hrs**
- Grading system
  
- **Barium contrast study**
- Not reliable in predicting the acute injury or the risk for stricture formation → not reliable early in the course
- Ideally, ***after 1-3 weeks*** of the significant injury

# Grading for caustic esophageal burn

Injury	Finding
Grade 1 (superficial)	Superficial erythema and edema
Grade (trans-mucosal)	Hemorrhage, exudates, linear erosions, blisters, shallow ulcers involving the mucosa and submucosa <u>Grade 2A</u> : Localized or partial <u>Grade 2 B</u> : Circumferential burn present
Grade 3	Deep ulceration, eschar formation with necrosis, full-thickness injury with and without perforation

→ Linear burns rarely lead to stenosis, whereas circumferential burns frequently heal with stricture formation

# Management



Depend on 2 important factors:  
1- Certainty of ingestion  
2- Presence of symptoms

# Medical management - General

- **ABC**
- **DO NOT DO 4- things:**
  - 1- Induce vomiting
  - 2- Using neutralizing agents
  - 3- Using dilution agents eg milk
  - 4- Trying to insert NGT blindly
- **NGT:** In patients with extensive circumferential burns (Grade 2B or 3) under direct visualization during EGD.
- PPI to prevent stress ulcers

# Is there a role for Steroid ?

- ***Animal studies & numerous small case series*** suggested a benefit in patients with first-or second-degree esophageal burns in preventing esophageal scarring
- A benefit of using corticosteroids in patients with third-degree burns has *not been demonstrated* (*inevitable stricture formation , may mask perforation*)

# Is there a role for Steroid ?

- Other studies → no benefit (and possible harm)
- Strictures developed in 20 to 38 % of documented esophageal burns, regardless of treatment.
- The largest RCT to address the issue included 60 children over an 18-year period who were assigned randomly to receive a placebo or prednisolone (2 mg/kg per day IV) followed by oral prednisone to complete a 3 weeks course
- Stricture formation appeared to be related only to the severity of the caustic injury, not the use of prednisolone.

Abstract

# Is there a role for Steroid ?

- A 2005 review of the value of corticosteroids examined reports on caustic ingestion between 1991 and 2004.
- There were 10 studies that met their criteria for review (2<sup>nd</sup>/3<sup>rd</sup> degree of esophageal injury, steroid used at least for 8 days or no steroid).
- This included a total of 572 patients; 8 of the studies included children.
- Conclusion: There did not appear to be any benefit in using corticosteroids for Grade 2 or 3 burns.



# Steroid and acid ingestion

- Corticosteroids **should not** be used for **acid ingestion** (esophageal strictures are less likely and there is a greater risk of masking the clinical features of gastric necrosis and perforation)

Hawkins, DB, Demeter, MJ, Barnett, TE. Laryngoscope 1980; 90:98

# When to start feeding?

- Patients with **grade 1 or 2A** injury require no specific therapy. A liquid diet may be initiated and the patient can be advanced to a regular diet in 24 to 48 hours.
- Patients with **grade 2B or 3** injury should have NG feeding initiated after 24 hours.

# Endoscopic management

- **Prophylactic dilatation** (before stricture formation):
  - does not eliminate stricture formation completely but the stricture can resolve more easily with early dilation in compare to patients who have dilation started after stricture formation
  - May increase risk of perforation → limit its use

Pediatr Surg Int. 2005 Feb;21(2):78-80

- **Late dilatation** after stricture formation
- **Mitomycin C**

# Mitomycin C

- It is an inhibitor of fibroblast proliferation
- It has been used in children who have required repeated dilatations
- No controlled trials
- Case series showed that the application of mitomycin C to the surface of the stenotic esophagus right after dilatation has been reported to decrease the need for further dilatation (62 % major success & 19 % partial success).

Uhlen S et al, Endoscopy. 2006 Apr;38(4):404-7

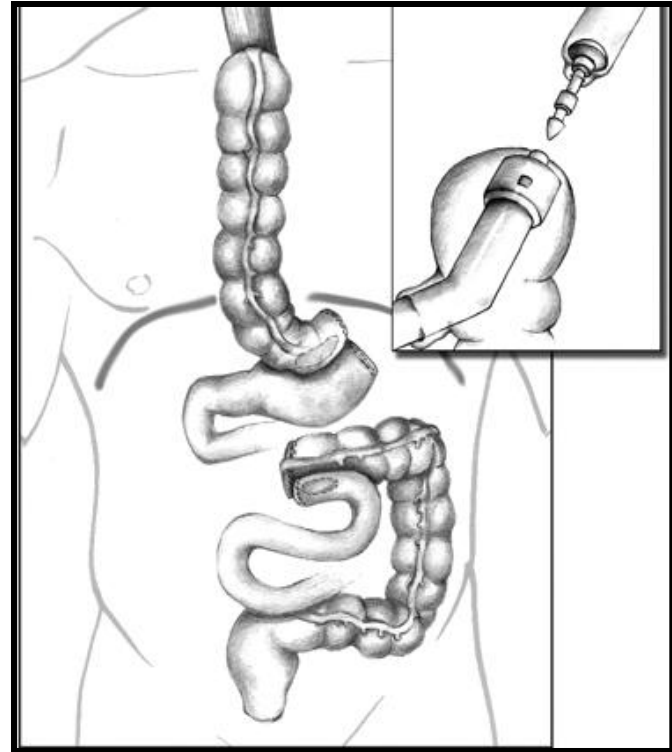
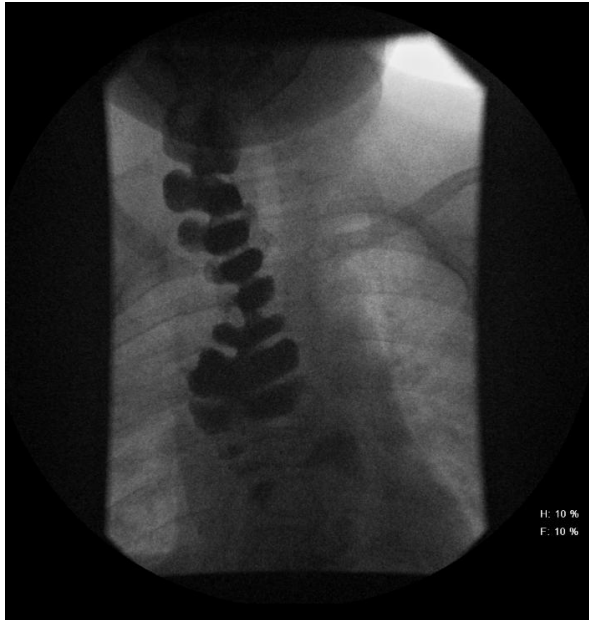
Rosseneu S et al, J Pediatr Gastroenterol Nutr. 2007 Mar;44(3):336-41.

# Surgical management

- ***Gastrostomy tubes:*** for sever & extensive injuries (if prolonged healing is expected)
- ***Esophagectomy with organ replacement*** (colonic, gastric or jejunum): may be required for patients with severe strictures not responding to medical therapy

# Surgical management

- Good prognostic factors if :  
good vascular supply and  
absence of tension at the  
anastomosis



# Surgical management

- Ideally, the replacement organ for esophagus should:
  - 1- be its luminal diameter is similar to that of the esophagus
  - 2- It has own peristaltic activity.
  - 3- This peristalsis follows (or is followed by) the peristaltic contraction of the previous (or oncoming) organ
  - 4- It has limited own secretion.
- Unfortunately, none of the currently used organs has these features → most of the patients have symptoms (dysphagia, regurgitation, chest pain, post-prandial fullness)

# Surgical management

- Anastomotic strictures may need periodic dilatation, and reflux of gastric contents can produce gastrocolic ulceration
- After exclusion of any structural defects for the symptoms, symptoms should be attributed to ***functional disturbances***.

Disease of the Esophagus (2003), 16; 291-294



## **Prokinetic effect of cisapride on pedicled stomach, small bowel and colon grafts replacing the esophagus after esophageal resection**

K. Kalmár,<sup>1</sup> Vereczkei,<sup>1</sup> K. Zámbó,<sup>2</sup> L. Pótó,<sup>3</sup> Ö. P. Horváth<sup>1</sup>

- Between 1995 and 1998, 15 adult patients with gastric pull up, 10 patients with colon replacement or bypass and 8 patients with free jejunum transplant or jejunum replacement were evaluated
- Esophageal transit scintigraphy was performed before and after cisapride administration.
- Cisapride exerts a good prokinetic effect on pedicled stomach and jejunum substitutes after esophageal resection, however, the results dispersed too widely to yield significant difference for the colonic interposition procedure.

# Late sequelae

- Esophageal stricture
- Esophageal dysmotility
- Esophageal Ca

# Esophageal stricture

- Esophageal Stricture formation occur in 3 to 57 % of ingestions with documented esophageal burns.
- Most third-degree (circumferential) burns lead to esophageal strictures, regardless of treatment
- Approximately 80 % of patients who develop a stricture will have obstructive symptoms within two months (earlier for the more sever injury).

Moazam, F, et al. South Med J 1987; 80:187.

Gaudreault, P, et al. Pediatrics 1983; 71:767.

Esophageal stricture dilatation

Andrea topic next week

# Esophageal stricture

- Only **33 to 48 %** of patients with caustic strictures have long-term success with ***repeated dilatations***.
- The remaining patients have usually extensive strictures that ultimately require ***esophagectomy with colon interposition within two years following the ingestion***.

Moazam, F, et al. Caustic South Med J 1987; 80:187.  
Anderson, KD, et al. N Engl J Med 1990; 323:637.

# Esophageal Ca

- Following colonic interposition, caustic injury appears to lead to an increased risk of carcinoma in the bypassed esophagus 1000-3000 times risk in compare to GP
- The time interval between ingestion and the detection of tumor has ranged from 16 to 42 years
- Late development of carcinoma is also seen in in patients who have a stricture but have not needed a colonic interposition.

# Esophageal Ca- Screening

- ***ASGE recommendations for surveillance:***
  - It should begin 15 to 20 years after the caustic ingestion.
  - The time interval of endoscopic surveillance requires study (generally, endoscopic examination should not be conducted more frequently than every 1 to 3 years).
  - There should be a low threshold to evaluate swallowing problems with endoscopy

The EnD