Caustic Injuries of the Esophagus

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May 10th 2012
Case Presentation

• **CC:** esophageal stricture

• **HPI:** This is a 31 y/o male who at age 16 ingested lye and underwent an esophagectomy with colonic interposition. He subsequently developed a stricture at the proximal anastomosis which led to intermittent difficulty tolerating a diet. He has had multiple dilatations since his initial operation (last 12/2011) and has now been referred for surgical evaluation and possible intervention.
• **PMHx:** gastric ulcerations

• **PSHx:** esophagectomy with colonic interposition, PEG (1997), esophageal dilatations (1997 - 2011)

• **Allergies:** Penicillin

• **Meds:** lansoprazole

• **SHx:** denies smoking, IVDA, occasional ETOH

• **FHx:** non-contributory
• Radiologic Studies: pre-op

CT Neck/Chest/Abd/Pelvis: s/p esophagectomy with colonic interposition, proximal anastomosis visualized without evidence of leak.

Esophagogram: tight smooth stricture approximately 2mm in diameter and 1cm in length at the proximal anastomosis
• Vitals: Temp 98.5°F  BP 120/75  HR 96  RR 18

• Physical Exam:
  
  General: AAOx3
  HEENT: left neck scar
  Chest: equal air entry bilaterally
  CVS: S1S2, rrr
  Abdomen: midline scar well healed
  LE: no edema
• Labs:
  CBC: 4.46 / 10.3 / 36.4 / 898

  Chem: 137 / 4.9 / 101 / 32 / 13 / 0.84 / 86

  LFTs: 7.9 / 4.5 / 16 / 13 / 83 / 0.4

  Coags: 12.0 / 27.9 / 1.0

Type & Screen: AB+

EKG    NSR    CXR    postsurgical changes
• **Intra-op:**
  - esophagoscopy
  - left neck exploration
  - resection of the esophago-colonic anastomosis (striction) with primary repair
  - JP placement x 2

• **Post-op:**
  - POD # 3-4 NGT removed; enteral feeds via PEG
  - POD # 6 neck incision opened and approximately 3cc purulent fluid drained; pt started on ice chips; clear fluid drained from JP

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• Post-op: cont’d

POD # 7 esophagogram: small to moderate leak
Post-op: cont’d

POD # 8-10 minimal output from JP
POD # 11 clear liquid diet
POD # 15 patient discharged home with JPs in place

Currently patient is tolerating a diet (solid and liquid)
Approximately 34,000 caustic injuries occur annually in the United States.

Leading cause of toxic exposure in children (accidental).

Second most common toxic ingestion in adults (suicidal attempts).
Caustic Injury to Esophagus

- Type of caustic injury:
  - Alkaline
  - Acid

- Mechanism of injury is based on the ingested chemical, concentration (strength), quantity and duration of contact
Mortalities are associated with ingestion of a strong alkali

- liquid lye
- solid clinitest tablets
- button batteries

- most concentrated bases available
- significant injuries to tissue occur at higher pH ≥ 11
Alkaline ingestion: high viscosity substances with slow transit time leading to prolonged exposure and deep tissue penetration causing *liquefactive necrosis*.

Acid ingestion: less viscous, fast transit time, limits tissue penetration and damage due to eschar formation, causes *coagulation necrosis*.
**Common Chemical Exposures**

<table>
<thead>
<tr>
<th>Chemicals</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Alkaline</strong></td>
<td></td>
</tr>
<tr>
<td>Sodium hypochlorite (weak base)</td>
<td>Bleaches</td>
</tr>
<tr>
<td>Ammonia Base (weak base)</td>
<td>Toilet bowl cleaners, hair dyes, floor strippers, glass cleaners</td>
</tr>
<tr>
<td>Sodium hydroxide (weak base)</td>
<td>Clinitest tablets, detergents, laundry powders, paint removers, drain cleaners, button batteries</td>
</tr>
<tr>
<td>Sodium borates, carbonates, phosphates</td>
<td>Detergents, electric dishwashers, water softeners</td>
</tr>
<tr>
<td><strong>Acid</strong></td>
<td></td>
</tr>
<tr>
<td>Hydrochloric acid</td>
<td>Swimming pool cleaners, metal and toilet bowl cleaners</td>
</tr>
<tr>
<td>Hydrofluoric acid</td>
<td>Antirust products</td>
</tr>
<tr>
<td>Sulfuric acid</td>
<td>Car battery fluids</td>
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</tbody>
</table>
Caustic Injury to Esophagus

Presentation

• Signs and Symptoms

- oropharyngeal pain
- odynophagia or dysphagia
- salivation
- chest pain
- stridor/hoarseness – laryngeal edema (orotracheal intubation)
- hematemesis – sloughing of esophageal mucosa
Presentation

- Signs and Symptoms: cont’d

  retrosternal pain radiating to back, acute epigastric pain — full thickness injury, possible esophageal perforation and impending mediastinitis

  massive ingestion - worsening hemodynamic instability or systemic signs (fever, tachycardia, mental status changes)

  clinical symptoms are poor predictors of the degree of injury
Caustic Injury to Esophagus

- **Presentation**
  
  significant tissue injury is usually noted within 24 hours

  Phase I injury: seconds, erythema and edema, superficial ulcers

  Phase II injury: progression of injury, vascular thrombosis with necrosis and mucosal sloughing (2-4 days) followed by granulation (3-4 weeks)

  *critical period: esophageal wall weakness*

  Phase III injury: fibroblast proliferation, cicatrisation, and stricture formation (weeks to months)
Management

• Key is to accurately identify the degree of injury and minimize its extension

• Airway assessment should be initially performed along with ATLS protocol
  - stridor (laryngeal injury) requires intubation
  - laryngoscopic exam: extent of injury

• NPO, IVF

• CXR, AXR: pneumomediastinum, pneumoperitoneum
  - full thickness injury; immediate operative intervention
Management

- Contraindications
  - blind passage of NGT or nasopharyngeal intubation
  - steps to dilute or neutralize the corrosive agent
    (neutralization of an acid or alkali can lead to an exothermic reaction thus further injuring the surrounding tissue)
  - induction of emesis: re-exposure to the esophagus
Caustic Injury to Esophagus

Management

- Endoscopy is gold standard
- Should occur within first 12-24 hours
- Indications: stridor, suicidal attempts, symptomatic children (vomiting, drooling), oropharyngeal burns
- Most perforations occur at day 2-3 due to friable granulation tissue therefore early endoscopy is mandated
### Management: Endoscopy

#### Classification of Injury

<table>
<thead>
<tr>
<th>Grade</th>
<th>Degree of Injury</th>
<th>Endoscopic Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Superficial mucosal burn</td>
<td>Mucosal edema and hyperemia</td>
</tr>
<tr>
<td>IIA</td>
<td>Transmucosal injury</td>
<td>Patchy ulcerations, exudates, sloughing mucosa</td>
</tr>
<tr>
<td>IIB</td>
<td>Transmucosal injury</td>
<td>Circumferential injury</td>
</tr>
<tr>
<td>III</td>
<td>Transmural injury</td>
<td>Deep ulcerations</td>
</tr>
<tr>
<td></td>
<td>+ Periesophageal or perigastric extension</td>
<td>Black or grey discoloration, full-thickness necrosis</td>
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Management: Endoscopy

- Grade of mucosal injury is the best predictor of complications
  - I, IIA no strictures
  - 70% IIB and all III strictures

- Limitations
  - unrecognized progression of injury
  - circumferential burn preventing full visualization
  - difficulty in distinguishing between grade IIB and III
Treatment

- Grade I and IIA (mild injury) – liquid diet, advanced to regular within 24-48 hours, contrast study to ensure no stricture in 3 weeks

- Grade IIB and III (severe injury) – observed for a minimum of 48 hours to 1 week, hyper-ailmentation or enteral feeds for nutrition, high index of suspicion for progression of injury, gastrostomy for nutrition and retrograde dilatations

- Prophylactic antibiotics are recommended; H 2 blockers or PPI for reduction of acid reflux; use of corticosteroids for stricture prevention is controversial
Treatment

• Surgical Intervention: patients with full thickness injury or evidence of perforation

• Timely operative intervention has decreased mortality from 20% to 3%

• Operative field prepped from the mandible to the pelvis - cervical, thoracic or abdominal approach
Treatment

- **Surgery performed is based on extent of injury**
  - esophagectomy, cervical esophagostomy and gastrostomy or jejunostomy
  - reconstruction via a transhiatal approach: gastric transposition or a substernal colonic interposition
  - colonic interposition: most widely used anatomic reconstruction similar anatomy; dependable blood supply
  - gastric tubes may be of inadequate length due to original injury and/or stricture or may not be used due to gastrostomy tube placement, dumping symptoms and reflux esophagitis
• Treatment Algorithm

![Caustic Ingestion Algorithm](image-url)

- Toxic Ingestion
- Evaluation ID Agent/Amount
  - Asymptomatic
    - Observe 24°
    - Follow-up Evaluation to ensure no loss of swallowing function
  - Symptoms: Pain, Dysphasia, Drooling and Oral Burns
  - Massive Ingestion, Suicidal Ingestion, Ingestion of concentrated agents
    - Airway Evaluation ATLS CXR/AXR
    - Evidence of Perforation
      - No
      - Yes
        - Early Endoscopy
          - Mild Injury
            - Abx
            - H₂ Blockers
          - Moderate/Severe Injury
            - Abx
            - H₂ Blockers
            - TPN/Enteral feedings
            - Gastrografin swallow +/- Endoscopy (D21)
            - Dilation of Stricture
        - Emergent surgery
Late Sequelae

- Major complication: stricture
- Worsening dysphagia usually implicates stricture formation
- Potential malignant transformation, increased 1000 fold
- Management:
  - dilatation (46-50Fr dilator in adults; 32-36Fr in children)
  - intractable stricture requires resection or esophagoplasty
Conclusions

• Management of caustic ingestion require radiologic and endoscopic evaluation to assess injury

• Mild injuries (I, IIA) are observed
  Severe injuries (IIB, III) without perforation are managed with supportive care, gastrostomy placement and surveillance for stricture formation

• Caustic perforation mandates immediate esophageal and/or gastric resection
References

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