Caustic injuries following high concentration peroxide ingestions: 2001-2011
Benjamin W. Hatten, L. Keith French, Robert G. Hendrickson, B. Zane Horowitz
Oregon Poison Center, Department of Emergency Medicine, Oregon Health & Science University

Background:
Caustic injury and hollow viscous perforation have been suggested as a consequence of high concentration peroxide ingestions.

Objectives:
In symptomatic peroxide ingestions of >10% concentration, to examine the role of esophagogastroduodenoscopy (EGD) in the diagnosis and treatment of caustic injuries and to examine the incidence of perforation.

Methods:
NPDS was queried for ingestions from 2001–2011 coded as a peroxide product with concentration >10%, hyperbaric oxygen (HBO) as a treatment, or an outcome code of moderate effects, major effects, or death. Charts were then obtained from all 57 open and 3/6 closed US Poison Control Centers.

Results:
After elimination of low concentration peroxide ingestions, 294 cases of symptomatic high concentration peroxide ingestion were available for analysis. 130 (44.2%) underwent an EGD, with a median time to EGD of 15 hours post ingestion.

- EGD was performed in 118/263 (44.6%) of those with emesis and 12/31 (38.7%) without emesis (p = 0.57).
- EGD was performed in 62/119 (52.1%) of those with hematemesis.

EGD results recorded for 118 patients

<table>
<thead>
<tr>
<th>Injury grading</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>normal</td>
<td>11.9%</td>
</tr>
<tr>
<td>grade 1</td>
<td>29.7%</td>
</tr>
<tr>
<td>grade 2a (superficial ulcers/erosions)</td>
<td>54.2%</td>
</tr>
<tr>
<td>grade 2b (deep ulcers)</td>
<td>2.5%</td>
</tr>
<tr>
<td>grade 3 (necrosis)</td>
<td>1.7%</td>
</tr>
<tr>
<td>5/118 (4.2% CI 1.4–9.6%)</td>
<td>exhibiting grade 2b or 3 lesions</td>
</tr>
</tbody>
</table>

One patient who underwent an EGD died. All 5 patients with grade 2b or 3 lesions presented with vomiting, 4/5 (80%) reported hematemesis. The age range for those with a grade 2b or 3 lesion was 42–87 years. The range of volume of peroxide ingested was 15–60 mL in those with a grade 2b or 3 lesions.

3/13 (23.1%) patients with embolic events who had an EGD experienced a grade 2b or 3 lesion compared to 2/103 (1.9%) of those without embolic symptoms who had an EGD (p=0.009).

All patients with grade 2b or 3 findings would be captured by restricting EGD to those with evidence of embolic symptoms. Significant GI bleed (melena or massive hematemesis), pneumomediastinum, or history of gastric bypass.

This would have reduced the number of patients undergoing EGD by two-thirds to 46/294 (15.6%) of patients.

2/294 (0.7%) had possible hollow viscus perforations.
Both were diagnosed as possible esophageal tears with pneumomediastinum by chest CT. Neither required surgical intervention.
In both cases, it was unclear if the injury was due to vomiting or the caustic properties of peroxide.

Conclusion:
Significant findings occurred in only 4.2% of concentrated peroxide ingestions who had grade 2b or 3 lesions.
All patients with grade 2b or 3 findings would be captured by restricting EGD to those with evidence of embolic symptoms, significant GI bleed, pneumomediastinum, or history of gastric bypass.
Possible perforation is very rare and can likely be diagnosed by CT.