Acids and Alkalis

Sources

- Acids and alkalis are also sometimes referred to as caustics or corrosives. Other compounds, such as concentrated phenol, concentrated iodine and silver nitrate may also have a corrosive or caustic effect. Therefore the terms corrosive or caustic are not synonymous with acids or bases.
- The frequency of serious alkali ingestion is more common than serious acid ingestion.
- Alkalis include:
  - Sodium hypochlorite (Clorox and similar bleach solutions) - One of the more common agents ingested.
  - Lye, which is sodium or potassium hydroxide - One of the most common causes of serious toxicoses.
- Acids include:
  - Hydrochloric, sulfuric and (rarely) hydrofluoric acids. Muriatic acid is another name for hydrochloric acid. Naturally there are others, but they are less commonly encountered as toxicants by domestic animals.

Mechanisms of Action

- Alkalis.
  - Hyperthermic injury, documented following lye ingestion.
  - Saponification of fats (independent of hyperthermic effect).
  - Stricture formation secondary to esophageal ulceration is unlikely unless the pH reaches 12.5.
- Acids.
  - Desiccation (drying) and generation of heat.
  - Acids do not tend to penetrate as deeply as do bases, but they do cause coagulation necrosis.
- Location, frequency and severity of lesions depends upon:
  - Chemical properties of the particular compound.
  - Volume and concentration to which the animal is exposed.
  - Contact time with the tissues.
  - Presence of other ingesta in the gastrointestinal tract at the time of exposure.
  - Tonicity of the pyloric sphincter.

Toxicity

- Lye tends to penetrate deeply into tissues whereas sodium hypochlorite and acids do not.
Severity of injury is proportional to the concentration and the volume of the toxic solution.

Prolonged contact increases the probability of severe injury increasing the risk of perforation.

A full stomach is less vulnerable to corrosive injury than an empty one because of dilution of the caustic agent.

Pylorospasm, which can be induced by the caustic agent, increases the risk of local gastric injury.

Because dogs and cats lap up materials their exposure to caustics tends to be slower and less of a problem than in children who "drink" such materials in quantity.

Signs and Lesions

- Alkali.
  - Lye commonly injures the oropharynx and esophagus and may injure the stomach as well.
  - In human patients solid lye tends to lodge in the oropharynx or esophagus and tissue injury at the site of contact continues until perforation occurs unless the particles are removed by vomiting or passed into the stomach.
  - Also in humans, liquid lye often presents a different and even more serious problem than solid lye. Up to 3 times as many patients developed esophageal injury after ingesting liquid lye than those ingesting solid lye. Liquid lye seems to cause almost immediate damage and often injures the stomach as well as the esophagus.
  - The most common immediate complication of lye ingestion is an esophageal burn which occasionally goes on to result in perforation. Gastric perforation is unusual but it occurs more often in patients ingesting liquid lye.
  - In some patients, respiratory distress occurs secondary to soft tissue swellings of the larynx, epiglottis, vocal cords or as a result of tracheal aspiration.
  - The principle delayed complications are esophageal stricture and pyloric obstruction.
  - Few instances of sodium hypochlorite induced esophageal stricture are on record and, in at least some of these, the history of exposure is of questionable certainty.

- Acids.
  - In human patients, the stomach is most frequently injured from acid ingestion, and the pyloric antrum is the area most often affected.
  - Esophageal injury is comparatively infrequent, affecting 6 - 20% of patients (humans).
  - Acute complications may include gastric perforation, acute peritonitis and shock, requiring immediate surgical and medical intervention.
  - Delayed complications may include sloughing of the gastric mucosa, peritonitis, perforation and sepsis. In some animals a primary pyloric stenosis may emerge over weeks to months after ingestion.

Diagnosis

- A history of exposure and appropriate clinical signs are sufficient for a tentative, clinical diagnosis.
- If possible use pH paper to assess the pH of the toxicant in its original container. Commonly, pH papers with a range of 1 - 12 are available as are high range papers with a range of 12 - 14. Both should be used if they are available (obtain ahead of time or check with a local hospital emergency department).
- As with other toxicoses, it is important to determine the particular agent ingested, its concentration, the quantity ingested, the duration since ingestion, whether vomiting has occurred since that time and whether a diluent was given thereafter.
- Examinations of the oral, pharyngeal and esophageal mucosae are desirable. It is possible to have significant esophageal pathology in the absence of oral damage, but this would be the exception, rather than the rule.

Treatment

- The use of emesis and lavage are generally regarded as contraindicated in acid or alkali ingestions, because of the possibility of re-exposure of the damaged tissues of the esophagus, because of the risk of rupturing an already weakened stomach, and because of the possibility of aspiration.
- Activated charcoal and saline cathartics are not indicated for ingestions of acids or alkalis because: 1) activated charcoal is a poor adsorbent for acids or alkalis, 2) tissue injury occurs so fast that, by the time the animal reaches the facility and is administered the treatment, it would do no good and 3) activated charcoal may limit the ability to characterize any damage endoscopically.
- Corticosteroids are suggested for caustic ingestions in which there has been circumferential mucosal damage in order to limit fibroplasia and thereby minimize stricture formation. In order to be of benefit, the corticosteroid should be given within 48 hours of exposure to the caustic agent. However, care must be taken in selecting patients for corticosteroids use. The drugs are contraindicated in the presence of perforation because of masking of clinical signs and the possibility of reducing the success of surgical correction. Noncircumferential esophageal burns do not require steroid therapy. When used, the steroids are given for a period of several weeks.
- Although disputed by some authors, experimental evidence is in favor of the use of antibiotics when there is a history of perforation and prophylactically when steroids are given.
- Respiratory distress, although serious when it occurs, is very rarely encountered. If severe, endotracheal intubation should be used if the tube can be inserted without causing additional damage to the laryngeal area. If that cannot be avoided, tracheostomy may be
required.

- Aspiration into the respiratory tract is also infrequent, but in view of the gastric contents and irritation which may be present, extreme care should be taken to avoid this effect.
- Monitoring blood gases and appropriate measures to alleviate respiratory or circulatory embarrassment leading to anoxia or shock may be of benefit in such patients. These may include artificial respiration, oxygen, and more often, fluids, bicarbonate, and corrective electrolyte solutions.
- An analgesic may be indicated to alleviate pain.
- Survey X-rays may be taken of the thorax and abdomen.
- The use of diluents, especially, milk and water, are often recommended for ingestion of acids or bases although some authors feel that such steps are at best of questionable benefit unless the patient is treated within minutes of exposure.

**Alkalis.**

- Diluents are used with 2 goals in mind: 1) To remove solid alkali materials from the oropharyngeal and esophageal mucosae; and 2) To dilute the caustic material and decrease the degree of tissue damage.
- Acids, such as lemon juice, acetic acid or vinegar are **contraindicated** in alkali toxicoses, due to the resultant exothermic reaction and associated thermal injury to the mucosae.
- For solid lye ingestions, the use of milk is preferred over water but if not immediately available water should be administered.
- For liquid lye ingestions, the use of diluents is more controversial because of a study involving humans, in which such therapy was associated with subsequent vomiting. The author feared that the associated reexposure of the esophageal mucosa would be likely to increase the injury. In view of the relatively infrequent occurrence of esophageal perforation in poisoned dogs and cats, the use of a diluent may have a more favorable risk/benefit ratio in these species than in humans. Investigation of the prevalence of vomiting and secondary complications in animals ingesting liquid lye with and without the addition of a diluent are needed to make a definitive recommendations.

**Acids.**

- Diluents are not known to be of reliable benefit in acid ingestion and, again, there is concern as to the possible increased risk of vomiting. Nevertheless, some of the most commonly relied upon reference systems (e.g., Poisindex) for human toxicoses still recommend their use. The use of a diluent is therefore recommended for acid ingestions, especially if the ingestion has occurred minutes prior to presentation.
- Antacids, especially bicarbonate, are potentially contraindicated due to the release of heat and in the case of bicarbonate, gas bubbles as well.

### Additional Toxicants

<table>
<thead>
<tr>
<th>Specific Agents</th>
<th>Major Species</th>
<th>Usual Time of Onset</th>
<th>Usual Duration (if survives)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypochlorites (concentrated forms)</td>
<td>Small animals</td>
<td>Seconds to hours</td>
<td>Days; rarely lethal</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>Dogs</td>
<td>Minutes to hours</td>
<td>Hours to days; often lethal</td>
</tr>
<tr>
<td>Phenolics (concentrated phenol or cresol)</td>
<td>Dogs</td>
<td>(See Hepatotoxic Chemicals and Drugs)</td>
<td></td>
</tr>
<tr>
<td>Cholinesterase inhibitors</td>
<td>(See Inhibitors of Cholinesterase)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moldly red clover (salframine)</td>
<td>(See Muscarinic Agonists)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trichothecene Mycotoxins</td>
<td>(See Toxicants that Affect the Gastrointestinal Tract)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- Phenolics (See Coal Tar, etc. under Hepatotoxic Chemicals and Drugs)
- Formaldehyde
- Cholinesterase Inhibitors (See Inhibitors of Cholinesterase)
- Moldy Red Clover (Salframine) (See Toxicants with Muscarinic Effects but No Nicotinic Effects)
- Trichothecenes (the more toxic members of the group can cause oral lesions) (See Toxicants that Affect the Gastrointestinal Tract)

All rights reserved. This document is available on-line at www.ivis.org. Document No. A2637.0899.